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NUTRITION IN PREGNANCY

The Effects of Dietary Deficiency in Pregnancy and the Detection and Treatment of Nutritional Deficiency Diseases

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NUTRITIVE failure is more likely to occur during the course of pregnancy and lactation than at any other period of a woman's life. The increments that these processes add to the nutritional requirements are such that without judicious regulation of the dietary some degree of deficiency occurs in many women regardless of the economic status. During reproduction, nutrients must be supplied not only to sustain the usual metabolic needs of the maternal organism but also to provide the building materials and to support and catalyze the building processes in embryonic differentiation and development. These needs are augmented by the preparations for lactation manifested by growth and differentiation of mammary tissue and by apparent storage of specific nutrients for this particular function. A considerable amount of investigation in the past decade has yielded positive information about the nutritional needs in these critical periods and about the diseases and complications which may occur as a result of nutritional deficiency. Strangely enough, there has been no demonstration that malnutrition has anything to do with conception in women. The pauper, who is more likely to have deficiency disease, has always been rich in children. At the Nutrition Clinic in Birmingham we find few exceptions to this idea; the poorer the mother, the larger her family.

The same factors that operate to induce nutritive failure in any person are applicable in pregnancy. Those commonly accepted are (1) failure of ingestion, (2) increased requirements, (3) interference with absorption, and (4) interference with utilization. Theoretically possible are the factors of (5) increased excretion and (6) increased destruction. Although all of these factors may show their influence during pregnancy, the failure of ingestion and the increased requirements account for most of the nutritional difficulties which arise. Recognition of the increased metabolic demands, particularly during the latter half of pregnancy and during lactation, is shown in the minimum dietary requirements established by the Food and Nutrition Board of the National Research Council in 1941 (9) (Table I). The figures representing the requirements of the nonpregnant woman are the minimum for the active woman. In every

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TABLE I.—RECOMMENDED DAILY ALLOWANCES OF SPECIFIC NUTRIENTS FOR THE NONPREGNANT, PREGNANT, AND LACTATING WOMAN*

Nutrients	Nonpregnant	Pregnant (latter period)	Lactating
Calories	2500	2500	3000
Protein (gm.)	60	85	100
Calcium (mgm.)	0.8	1.5	2.0
Iron (mgm.)	12	15	15
Vitamin A (I.U.)	5000	6000	8000
Thiamine (mgm.)	1.5	1.8	2.3
Ascorbic acid (mgm.)	70	100	150
Riboflavin (mgm.)	2.2	2.5	3.0
Nicotinic acid (mgm.)	15	18	23
Vitamin D (I.U.)	+	400–800	400–800

*Food and Nutrition Board, National Research Council, Reprint and Circular Series No. 115, 1941.

instance pregnancy or lactation increases the minimum values. The nonpregnant woman whose diet just meets the requirements for her state of activity will have to alter her eating program to be efficient in reproduction. It is obvious, too, that the tissues of the woman who is on a dietary of borderline adequacy will become deficient when she becomes pregnant if her eating habits are unchanged. The demands of the fetus are preferential and progress geometrically, and as a result signs and symptoms of nutritional disturbances are commonly observed in the latter half of pregnancy.

The fetus fulfills its requirements not only from the raw materials and maternal stores but also is able to drain the maternal tissues of required materials. Therefore, the mother, rather than the fetus, tends to show the obvious marks of nutritive failure. The newborn, however, does not entirely escape the stigmas of the mother's poor nutrition, as is so aptly shown in a recent comprehensive review by Warkany (59).

A multitude of circumstances beyond the parasitic demands of the fetus predisposes to nutritional disease in the mother. The economic status of the family naturally influences the quality and sometimes the quantity of the dietary. The availability of protein, vitamins A, B_1, B_2, and C increases with rising increments of income (65), but availability does not insure an adequate dietary. Irrespective of income, the food consumed by many women during pregnancy tends to fall short of the optimum. Williams and Fralin (65), assaying the diets of 514 pregnant women, found only 2 per cent which met the recommended requirements. Forty per cent were fair, and 57 per cent were poor. Arnell, Goldman, and Bertucci (1) studied the dietaries of 400 unselected pregnant women and found 80 per cent only fair or less than fair in protein intake. Burke, Harding, and Stuart (8), evaluating only the protein content of the diets of 216 women, found only 10 per cent adequate in this nutrient. The mild nausea and vomiting of the first trimester and the peculiar food idiosyncrasies that appear at this time usually have no immediate clinical effect but may inaugurate erroneous dietary habits for the rest of the term. Proper dietary education during the early part of pregnancy can, therefore, be of tremendous prophylactic value. The size of the family bears a relationship to the mother's nutritional status. This is true particularly of the woman who is pregnant most of the time during her fertile years. It is a common clinical observation that signs of nutritive failure are more likely to appear in the woman carrying her sixth or seventh child than in the woman carrying her first or second child.

A study of 229 mothers between the ages of 18 and 45 at the Nutrition Clinic in Birmingham was made in relation to deficiency disease symptoms to the age, to the time of their appearance, and to the number of pregnancies. After gathering such material, however, it was obvious that statistical interpretation would prove fallacious. The group of unmarried women of comparable age who came to the clinic was too small for statistical analysis. The history of the first signs and symptoms was difficult to interpret and often too ephemoral for us to establish as definitely due to nutritional disturbances. In the groups of women who had 8 to 13 pregnancies the numbers were too few for analysis. It was interesting to find, however, that only 4 of the 22 mothers had definite symptoms of nutritional disease prior to the first pregnancy. One hun

dred thirty-five developed symptoms during lactation or pregnancy; symptoms appeared most frequently during the third trimester. Although the greatest number of women were in the groups which had 4, 5, and 6 pregnancies, the incidence of onset of symptoms was highest during the 4th and 5th pregnancies. Ninety of the women developed symptoms only 3 or more years after the last pregnancy.

In the lower economic brackets, repeated pregnancies introduce a vicious cycle of suboptimal nutrition. The allotment of food for the family remains essentially unchanged as the demand for it increases. The mother, moreover, is likely to give the food of higher quality to her husband and children. Such factors may well account for the predominance of women over men seen in the Nutrition Clinic in Birmingham (47). Hyperemesis gravidarum is more than a predisposing factor to nutritive failure; if it persists, it usually induces some manifestations of nutritional disease (34), particularly pellagra, scurvy, polyneuritis, and nutritional anemia. Signs and symptoms of the deficiency diseases are more likely to appear in the last trimester or during lactation, in a woman who has already had several children and whose dietary has been reduced by poverty or conditioned by erroneous food habits.

A number of investigations have indicated the general relationship of the state of nutrition to untoward events during pregnancy and lactation and to the health of the newborn. Comparing the offspring up to 6 months of age of women on adequate and on deficient diets, Ebbs, Brown, and Tisdall (11) have shown not only a higher morbidity but also a higher mortality in infants of mothers on poor diets. Supplementing the poor diet in 100 of the women in the group studied, in order to make the dietary approach the requirements, resulted in striking contrasts. As compared with the pregnant women who remained on the poor diets which yielded 1800 calories at the most, the women whose diets had been reinforced showed a natural gain in appetite so that the caloric intake rose to 2550 calories with a concomitant increased intake of the essential nutrients (12). Their interest and morale were improved. Only 6 per cent of these, as compared with 34 per cent of those women on the poor diet, were given a poor prenatal rating by the obstetrician. The other conditions during labor and the puerperium were manifestly bettered. The incidence of miscarriages, stillbirths, premature births, and of all complications of pregnancy was significantly lower in the women whose nutrition had been improved. It is notable that none of the women on either dietary showed clinical signs of any specific nutritional deficiency. The deficit was "preclinical" or biochemical. Balfour observed similar evidence of the effects of the nutritional status. Supplements mainly of vitamins A, D, and B complex, calcium, phosphorus, and iron were given to 11,618 pregnant women. There were 8,095 pregnant women who served as controls. All the women were in the low wage earning class. The stillbirth and neonatal mortality rates were considerably lower in the group of women on reinforced dietaries, but no evaluation was made of the general complications of pregnancy. Conversely, Williams and Fralin (65), working with assessment of dietaries, were unable to correlate deficiency of vitamins A, C, B_1, or of protein with the incidence of prenatal or postpartum accidents, with toxemia, with inadequate lactation, and with purely medical complications of pregnancy. Dieckmann, who studied the combined effects of calcium, phosphorus, iron, and vitamins A and D, recognized no advantages for mother or infant when these nutrients complemented the dietary in pregnancy. The majority of investigations, however, of the effects of specific nutrients strongly confirms the beneficial results incurred by the mother consuming an adequate diet.

As nutritive failure progresses in the last months of pregnancy, certain signs and symptoms appear which might lead to a diagnosis of anemia or of pellagra or neuritis, or another single deficiency syndrome. These isolated syndromes, however, are always a manifestation of an overall and mixed nutritional deficiency. Although iron deficiency anemia may exist alone because of chronic blood loss, a macrocytic anemia, due to deficiency of antianemic principles found in animal protein, implies also a protein and niacin deficiency.

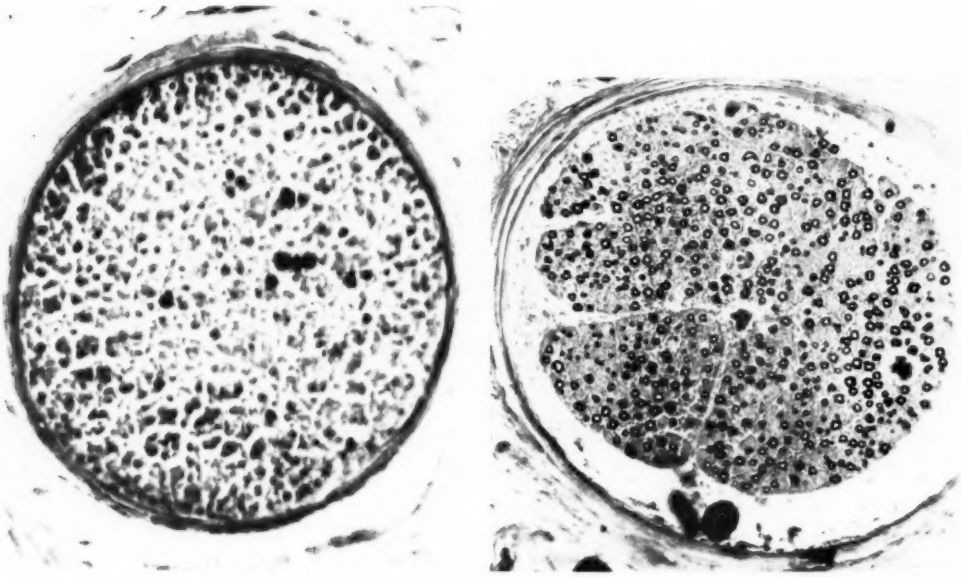

Fig. 1. Left, section of a branch of anterior tibial nerve of a 27 year old woman with peripheral neuritis due to thiamine deficiency. The myelin sheath count is 380 per square millimeter. (Osmic acid stain, ×225.) Compare with similar section from normal person, right, myelin sheath count 6836 per square millimeter. (Osmic acid stain, ×150.)

The significance, then, of the appearance of pellagrous glossitis lies not so much in the fact that the individual has pellagra or niacin deficiency, but in the certain implication that there is a biochemical deficiency of the entire B complex and probably of protein and of the other essential nutrients. Treatment of the single symptom with a single specific substance and omission of application of dietary control and combined therapy results in incomplete recovery.

Certain symptoms recognized as complications of pregnancy long before the unraveling of the B complex was undertaken are recognized now as deficiency syndromes. Such are polyneuritis of pregnancy and so called pernicious anemia of pregnancy. Polyneuritis of pregnancy is actually beriberi. If the dietary contains an adequate amount of thiamine, it does not develop. Synthetic thiamine in doses of 5 to 20 milligrams daily ameliorates the disorder. The signs and symptoms are familiar; they frequently follow hyperemesis gravidarum and hence may appear early in pregnancy. Initially there is generalized weakness and malaise followed by tingling and burning in the feet and later by other paresthesias. There is tenderness of the calf muscles, hyperesthesia on stroking the skin with an applicator, and tenderness on tapping the soles. The reflexes may be hyperactive (early), hypoactive, or absent (late). Foot drop may develop later on as a manifestation of peripheral motor involvement. Atrophy of the muscles and skin may be evident along with signs of vasomotor irregularities such as excessive sweating and the tendency for cyanotic mottling of the dependent extremities. The neuritis is of a stocking distribution and usually symmetrical. The upper extremities commonly are involved with progression of the disorder. Pathologic alterations (Fig. 1) correlated with these symptoms are swelling and degeneration of the myelin sheaths of the peripheral nerves. Other signs of thiamine and B complex deficiency such as anorexia and the "initial nervous syndrome" are common long before neuritis makes its appearance. Beriberi, heart disease, and encephalopathy can occur, but according to recent reports these

Fig. 2.

Fig. 3.

Fig. 2. Pellagrous glossitis involving the tip of the tongue in a 35 year old mother in the sixth month of her seventh pregnancy. Her first attack of pellagra occurred while nursing her second child, and this was associated with cheilosis and symptoms of peripheral neuritis in the feet and legs. These symptoms recurred with each following pregnancy. Her diet consisted chiefly of dried beans, cornmeal, flour, potatoes, and salt pork.

Fig. 3. Cheilosis (angular stomatitis) in a 36 year old mother of eight children. All children had been breast fed for at least 2 years. She was nursing a 5 month old child at the time that the angles of the mouth became sore. The infant showed no diagnostic lesions. Within the period of her recall her diet had consisted of cornbread, biscuit, dried vegetables, fat pork, and desserts. Six milligrams of riboflavin given daily resulted in the healing of the cheilosis. Eighteen months later she was again pregnant, but her diet had not improved. During the sixth month the cheilosis recurred.

Nutrition in Pregnancy, the Effects of Dietary Deficiency in Pregnancy and the Detection and Treatment of Nutritional Deficiency Diseases.
Carl F. Vilter, Dorcas Morgan, and Tom D. Spies

UNIV. OF MICH.

are relatively rare in the United States. Nonspecific electrocardiographic changes which can be attributed to thiamine deficiency are too irregular, according to Williams, Griffith, and Fralin (66), to be of diagnostic significance.

The daily thiamine requirement during pregnancy has been put at 1.8 milligrams. This, however, is not an absolute figure. Thiamine excretion tests designed to interpret requirement and saturation of tissue in the latter months of pregnancy are not in agreement (28, 43, 54) except on the general principle that the requirement is increased.

Beriberi can occur in the newborn (39) but is quite unusual. Heart disease in the infant due to beriberi, however, is occasionally seen in this country (55). This condition may occur at any time during the first few years of life, and reflection is cast on the maternal nutrition as a cause.

The macrocytic anemia of pregnancy, frequently referred to as pernicious anemia of pregnancy, is not thoroughly understood. Morphologically it cannot be differentiated from the anemia of sprue, nutritional macrocytic anemia, or pernicious anemia. Clinically it resembles nutritional macrocytic anemia (38) and seems identical except for its occurrence during pregnancy and its occasional refractiveness to parenteral treatment with liver preparations. The usual story indicates that nausea, vomiting, or anorexia so affects the dietary that little food of any kind is ingested. The patient becomes pale and weak, frequently has a sore tongue or peripheral neuritis and sometimes edema. The red cell count often falls below a million, and the cells are usually large but only moderately filled with hemoglobin. The white cell count is normal. The bone marrow is characterized by megaloblastic arrest. In most instances there is normal gastric secretion. Reports from India, where the picture is much more common, indicate that parenteral liver extracts are often of no value and that oral liver preparations have been effective therapy. These observations have been confirmed (62) in this country. Synthetic folic acid (the Lactobacillus casei factor), a vitamin of the B complex, has recently proved effective in the treatment of all varieties of macrocytic anemia due to deficiency of liver principle (36, 46, 48, 57), not excepting the macrocytic anemia of pregnancy. Whether given orally or parenterally, an adequate daily dose (approximately 10 mgm.) is followed by appearance of reticulocytes in the peripheral blood and restoration of blood counts to normal. Since this is a relatively rare dyscrasia in the United States and the use of "folic acid" in treatment so recent, it remains to be seen whether certain patients are refractive also to this vitamin.

Niacin deficiency produces in the pregnant woman the same signs and symptoms of pellagra that are found under circumstances unrelated to reproduction. The "initial nervous syndrome" of pellagra probably is a sign of mixed B complex deficiency in which thiamine and niacin are of greatest importance. Nervousness, insomnia, irritability, noise intolerance, weakness, lassitude, constant headache, and many other symptoms commonly associated with neurasthenia constitute this picture (14). These symptoms may progress into hallucinatory, paranoid, and negativistic states which, even in nonpregnant women, resemble prenatal and postpartum psychoses. There is a possibility that such mental aberrations in pregnancy and the puerperium might be related to deficiency of B complex vitamins.

There is no need for the detailed description of the familiar signs and symptoms of advanced pellagra, the symmetrical skin lesions, the atrophic red burning tongue, the intermittent foul diarrhea, and the encephalopathy. It should suffice to point out that these signs and symptoms may occur singly or in any combination and tend to recur in spring and in fall. They are most likely to afflict a woman during the last half of pregnancy or during lactation (See Fig. 2). Although signs of pellagra have not been noted in the newborn, they are occasionally seen in the first 6 months of life. Pellagra in the nursing infant can be treated by giving niacinamide to the mother (45). It is apparent that essential nutrients given to the mother are concentrated in mother's milk for the benefit of the child. Even if the mother shows symptoms of pellagra, her needs remain in abeyance as the materials are excreted in the milk.

Riboflavin deficiency, characterized by angular stomatitis (cheilosis) and a nonspecific conjunctivitis, causes no really embarrassing difficulties for the pregnant woman (see Fig. 3). At least none are recognized at the present time. In view, however, of investigation on rats conducted by Warkany and his associates (58, 60), more attention should be turned to the effects of this vitamin deficiency. Warkany showed that deficiency of riboflavin during the differentiation stage of the rat embryo produced congenital abnormalities such as shortening or absence of ribs, digits, and extremities, shortening of the mandible, cleft palate, deformities of the eyes, and similar anomalies. Since differentiation of the human embryo is accomplished by the end of the third month, deficiency of riboflavin might be most serious during the first trimester. There has been no correlation of human congenital anomalies, however, with diets low in riboflavin. Cheilosis and sometimes ocular symptoms occasionally appear in the nursing infant. These, too, may be treated by treating the mother.

Other members of the B complex are of uncertain value in human nutrition. Certain of these have been proposed as useful in treating morning sickness and the even more severe forms of nausea and vomiting of pregnancy. Vitamin B_6, pyridoxine, is purported to be valuable in these instances. Several investigators (17, 63) using 50 to 2500 milligrams of pyridoxine daily, both orally and parenterally, have reported amelioration of symptoms in the majority of their patients. There has been no evidence, however, that such women are deficient in vitamin B_6. No saturation and excretion tests have been performed to this end. The picture of B_6 deficiency in man is unrecognized; and in view of the wide distribution of the vitamin in foods, there is reason to question whether this deficiency ever occurs. The authors (56), as well as many others, have noticed an inconstant, mildly palliative effect of pyridoxine on the anorexia and nausea of x-ray sickness and consider the results of such therapy to be due to a pharmacologic action of the substance. Perhaps it acts as a gastric sedative. The unpredictable soporific effect of parenterally administered vitamin B_6 is well known and may be another manifestation of its sedative action. So it would seem that if pyridoxine is of aid in nausea and vomiting of pregnancy it is not because a specific vitamin deficiency is responding to treatment.

The clinical manifestation of vitamin C deficiency, scurvy, rarely occurs with pregnancy. When scurvy does show itself, it is usually induced by protracted hyperemesis gravidarum in a patient whose reserves of vitamin C were low before pregnancy. Lund and Kimble (30) believe that retinal hemorrhages in hyperemesis are scorbutic in origin. An occasional case of postpartum hemorrhage can be attributed to this deficiency since in scurvy bleeding may occur in any organ or tissue. The more commonly recognized signs of scurvy, such as spongy purple bleeding gums, keratotic hair follicles with perifollicular hemorrhages, and extensive subcutaneous ecchymoses, may be apparent. As in the case of the other vitamins, the fetus obtains its vitamin C at the expense of the mother, and the fetal plasma vitamin C level is always greater than that of the mother. Depending upon the degree of deficiency in the mother, the fetal plasma level may be 1½ to 6 times as great (3c). The fetus apparently is efficient in monopolizing the maternal sources of ascorbic acid since, even in infants nursed by deficient mothers, scurvy (Barlow's disease) is unlikely to occur before the age of 6 months. Under circumstances of extreme maternal deficiency, however, infants have been born showing scorbutic changes (2c).

Deficiency of the fat soluble vitamins, excepting vitamin K, is seldom a damaging influence in pregnancy in the United States. Dietary deficiency of vitamin K probably does not exist. On the contrary, a nutritional deficiency due to inability to absorb the vitamin is commonly recognized under those conditions which prevent the normal flow of bile into the intestinal tract. The reason for vitamin K or prothrombin deficiency in the newborn is still not thoroughly understood. The now usual practice, however, of administering vitamin K parenterally to the mother an hour or so before delivery or to the infant upon delivery has almost eradicated hemorrhagic

disease of the newborn. This practice is to be recommended. It is so generally employed, however, an extensive review of evidence and the implications of evidence is superfluous.

Although vitamin A deficiency is manifested to a slight degree in the general population by night blindness (nyctalopia) and perhaps by skin changes such as hyperkeratosis of the hair follicles (phymaderma), there is no clinical evidence that the course or product of human pregnancy is embarrassed by a deficit of this nutrient. Lund and Kimble (31) have studied the blood levels of vitamin A during pregnancy and the puerperium and find that in spite of adequate diets there is a progressive fall in the plasma level as pregnancy approaches term. After delivery there is a sudden rise in the blood level. These authors consider the progressive fall during pregnancy an indication of storage and the rise in the early puerperium a mobilization directed toward lactation. This fall, however, also correlates with the moderate fall in plasma proteins and erythrocytes and hemoglobin usually attributed to the hydremia of pregnancy. Whether molecules smaller than the proteins or the formed elements would be influenced by the dilution factor, however, is not certain. The same authors found the rate of puerperal morbidity and the complications of pregnancy, excluding toxemia, to be lower in those women whose plasma levels for vitamin A remained near normal. This relationship probably correlates more closely with the general nutritional status.

Animal experiments can be cited to show what severe maternal vitamin A deficiency will do to the offspring. Congenital blindness is induced by this deficiency in the calf (37), pig (15), and rat (61). Using the rat as the experimental subject, Warkany and Schraffenberger found the blindness due to poor differentiation of the embryonic structures, connective tissue replacing many of the specialized tissues. Congenital cataracts and blindness in the human have not been related to aberrant vitamin A metabolism; but animal experiments have directed the discovery of such a relationship in many other instances.

Vitamin D deficiency in the pregnant woman is seldom recognized except for a rare case of osteomalacia and as a complication of sprue, but a certain minimum of vitamin D is necessary for the normal development of the fetus. The metabolism of vitamin D is intimately related to that of calcium, phosphorus, the parathormone, and the exposure to sunlight, and does not allow for brevity in a review. Fetal rickets does occur (26, 27, 32) but is extremely rare in the United States. Even with relatively poor maternal diets, the fetus usually draws an apparently adequate amount of calcium from maternal stores, without causing the mother to become osteoporotic. The mother's teeth, moreover, do not suffer from increased caries in pregnancy, irrespective of the degree of negative calcium balance. It is a physiological impossibility to drain calcium from the erupted tooth; so pregnancy does not aggravate or initiate dental caries (67). In moderate maternal calcium deficiency, there is likely to be poor calcification of the bony structures of the fetus and maldevelopment of the deciduous teeth. Burke (7) notes defective deciduous teeth in a large percentage of children of mothers who were getting one pint or less of milk daily during pregnancy. Finola, Trump, and Greenson (13) have reported that the blood calcium falls slightly, though not to abnormal levels, in the latter months of pregnancy and that supplements of dicalcium phosphate and vitamin D prevent this decrease. Such supplements increase the calcification of fetal bones; and although it has been suggested that this may prevent molding of the head, difficult labors from this cause have not been noticed (6). There may be some danger in the administration of an excess of vitamin D and calcium since some instances of extensive calcification of the placenta and of the fetal kidney have been attributed to such supplementation (6).

Studies on the effect of vitamin E in pregnancy, as well as in general human nutrition, have not yielded conclusive data. Vitamin E has been suggested as a preventive and therapeutic measure for abruptio placentae and threatened abortion (29, 42), but these observations have not been confirmed.

The most common form of true anemia occurring in pregnancy and in women in general is that due to iron deficiency and chronic blood

loss. Undoubtedly this anemia results from excessive loss of blood through menstruation, coupled with inadequate quantities of iron in the dietary. It is impossible to determine menstrual loss of blood through the history (49) and so to establish the primary etiology. This anemia is almost invariably of the microcytic hypochromic type and, depending on the degree, is manifest usually only by pallor, listlessness, and fatigue. In severe cases which have persisted for several years, other symptoms arise such as burning of the tongue and dysphagia and associated signs of hypochlorhydria or achlorhydria, atrophy of the papillae of the tongue, spoon nails, and sometimes a semilunar membrane partially obstructing the upper third of the esophagus. This type of anemia is common in pregnancy and responds readily to ferrous sulfate or ferrous gluconate, 1 gram daily given in divided doses. There is a distinct tendency toward relapse when therapy is discontinued, since the precipitating cause is often not eliminated until the woman reaches the menopause. Occasionally, as in the cases with achlorhydria where ionization is poor, the dose of iron must be doubled or trebled. Liver extract has no place in the treatment of this kind of anemia. The fetus is apparently unharmed by the mother's anemia, but the mother's general efficiency is reduced. There is probably no relationship between hypochromic anemia and toxemia (5), although this association has been suggested (21).

In recent years the importance of optimum protein in the dietary has impressed both obstetricians and nutritionists. There has been an ever increasing accumulation of evidence that deficiency of protein affects both mother and fetus adversely and that the maintenance of an adequate protein intake (85+ grams daily) is a major factor in directing an efficient pregnancy. As previously noted, Burke, Harding, and Stuart (8) found that, of 216 women whose diets were assayed, only 10 per cent were eating the optimum amounts of protein and that the majority of the remaining 90 per cent were far below this standard. Leverton and McMillan discovered that 58 grams was the average daily protein consumption in the group of pregnant women in their study, and other investigators have found similar suboptimal figures (1, 3, 11, 24, 65). Burke and coworkers (8) have shown an increase in birth length and birth weight of infants with each additional increment of protein in the maternal diet. The larger infants were not simply big; their pediatric ratings were much superior to the ratings of those whose mothers had low protein diets. The mothers on the low protein diets tended to have more difficult types of delivery; so the size of the infants did not enter into this consideration. Arnell and his coworkers (1), however, found no relationship between size of infant and state of maternal protein deficiency.

The plasma proteins, more specifically the albumin fraction, tend to fall during the course of the normal pregnancy (41), apparently because of the phenomenon of hydremia. On a low protein dietary, however, the fall, reflecting tissue protein content, is exaggerated; and if a certain critical level is reached, nutritional edema appears. This is similar to nutritional edema in nonpregnant states. Arnell and coworkers (2, 3) reported 8 such cases in pregnancy and noted the dramatic response in diuresis and weight loss following institution of a high protein diet supplemented by plasma, blood, and casein digests. The low protein dietary affects other aspects of nutrition. Hemoglobin is poorly formed when protein is inadequate (5) and the portion of available calcium which is absorbed is reduced concomitantly with a reduction of ingested protein (33). For more details on the aspects of protein nutrition in pregnancy, the reader is referred to the recent review by Williams (64).

The relation of the adequate diet to toxemia of pregnancy has been under investigation for a number of years. Suggestions that deficits of vitamins A, D, calcium (35, 52), and members of the vitamin B complex (23, 52) might be responsible for this syndrome have been discredited (22, 31, 43, 65). Protein deficiency, however, if not the initiating cause of toxemia, is a prominent predisposing factor. The school of thought that high protein intake induced toxemia has given way to the idea that the very opposite is true. Low plasma proteins have been associated with toxemia

with and without edema for many years (40). Strauss (50, 51) feels that nutritional deficiency is the most important etiologic agent in toxemia. A group of his patients with toxemia treated by a diet which yielded 260 grams protein, 150 grams carbohydrate, and 70 grams fat showed diuresis and weight loss, fall in blood pressure to normal, and cessation of headache and visual disturbances. This was accompanied by a rise in plasma proteins and colloid osmotic pressure within 2 weeks. A similar group treated with a low protein diet remained unchanged or grew worse. Harden, McElroy, and Huggins (16), reviewing a large series of toxemias, concluded that an adequate intake of protein was an important measure in the prophylaxis of this disorder. More recently Bibb (5) investigated the plasma proteins of a large group of women during the course of pregnancy and concluded (1) that women whose serum proteins remained normal throughout pregnancy did not become "toxic"; (2) that if the values averaged below 6 grams per cent during most of pregnancy toxemia is to be expected; and (3) that a low serum protein in the last trimester is often a warning of impending toxemia, especially if the protein has been normal earlier. Tompkins and Holmes have also produced statistical evidence to the effect that inadequate protein in the dietary predisposes to toxemia. On the other hand, none of the 8 patients with frank nutritional edema reported by Arnell and coworkers (2, 3) had toxemia. In the 4,004 pregnancies studied by Kooser among women whose dietaries included a median of 45 grams protein daily, the incidence of toxemia was only 3 per cent. In the winter and spring, when the dietary was poor in animal protein, toxic symptoms were more than twice as frequent as they were in the summer and fall. Arnell, Goldman, and Bertucci (1), however, have recently reported further evidence favoring the relationship of protein deficiency and toxemia. Some investigators still believe that there is no correlation between the amount of dietary protein and toxemia, but it is reasonably safe to say that the woman whose protein intake during pregnancy has been adequate usually does not develop toxemia. Such a dietary includes a quart of milk, two moderate servings of meat, and one to two eggs daily.

A good supply of protein during pregnancy also insures the best possibilities for successful lactation. It has been calculated that the positive nitrogen balance during the gestation period is greater than that prescribed by the demands of fetal development and maternal maintenance (19). This implies the storage of protein against the development of the breasts and milk production.

It is obvious to nutritionists that obesity and progressive and excessive weight gain during pregnancy do not imply an optimal nutritional status. It is not unusual to find florid signs of pellagra, beriberi, and similar disorders in obese people, even though the common experience is that the anorexia accompanying B complex deficiency curtails the intake of all food. The obstetrician objects to the excessive gain in weight because of the mechanical difficulties it may entail in delivery. After the delivery, the mother, instead of reverting to her state prior to pregnancy, the state which she expects, finds she has the remaining excess weight to contend with. The views of obstetricians and nutritionists parallel in the reasonable control of weight increase in pregnancy. Unwarranted gains can be obliterated by reduction of the fats and carbohydrates in the dietary without reduction of the essential nutrients. The daily intake of one quart of milk, two servings of meat, an egg, green and yellow vegetable, citrus fruits, and a whole grain cereal should be maintained. The avoidance, however, of candy, sugar, and concentrated carbohydrates such as cake and pastry, and the reduction of bread and potatoes do much to eliminate undesirable weight increase. Even more effective is the reduction of fats and fatty foods. Skimmed milk may be substituted for whole milk in some cases, but the total amount should not be reduced. Through such measures the dietary can be reduced from the caloric standpoint without disturbing a balance of essential nutrients.

The treatment of nutritive failure in pregnancy is identical with that offered for nutritive failure under any circumstances. Of first importance is a diet providing 3500 to 4000

calories including 120 to 150 grams protein and adequate vitamins and minerals. Synthetic vitamins in balanced amounts should be added, e.g., thiamine, 10 milligrams; riboflavin, 5 milligrams; niacin, 50 milligrams; ascorbic acid, 75 milligrams. Specific therapy should be directed toward the predominant symptoms. If pellagrous glossitis is present, the regimen is supplemented by 300 to 500 milligrams of niacin. If protein deficiency is predominant, the dietary protein is raised, arriving at 150 grams. This can be achieved by natural foods alone or by supplementing with protein hydrolysates. Often the physician finds it wise to provide some natural source of vitamin B complex such as brewer's yeast (60 gm.), crude liver extract, or wheat germ. Prophylaxis, of course, eliminates the entire problem of primary dietary deficiency disease, but this is difficult in the lower economic strata of most communities and impossible when the pregnancy comes under the surveillance of the physician only in the latter months.

SUMMARY AND CONCLUSIONS

1. Evidence from this study and from studies of others directs the attention of the physician to the importance of optimal nutrition during pregnancy. Illustrations of cases in pregnant women show: neuritis of thiamine deficiency in a 27 year old woman (Fig. 1); pellagrous glossitis in a 35 year old mother occurring in the last trimester of her seventh pregnancy (Fig. 2); riboflavin deficiency symptoms which developed in the mother of 8 children during a period of lactation (Fig. 3).

2. Methods for the prevention of nutritional deficiencies are presented and means of diagnosis are described. The principles of treatment advised for nutritional disease occurring during pregnancy do not differ from those prescribed in nonpregnant women, namely:

a. A diet providing 3500 to 4000 calories, including 120 to 150 grams protein, adequate vitamins and minerals.

b. Synthetic vitamins in balanced amounts.

c. Specific therapy with synthetic vitamins directed at the more prominent symptoms.

d. A natural source of vitamin B complex such as brewer's yeast, crude liver extract, or wheat germ.

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GASTRIC CARCINOMA: INCIDENCE AND DIAGNOSTIC PROCEDURES

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IN 1938, there were 150,000 deaths in the United States from cancer, 27,000 (approximately 18 per cent) being due to cancer of the stomach (2). In 1940 there were 156,000 cancer deaths (3). It is predicted that in 1950 there will be 203,000 such deaths and in 1960, 270,000. It is estimated that by 1980, 40 per cent of the population of the United States will be over 45 years of age. The incidence of this disease will undoubtedly continue to rise as the number of people in the older age group increases further. It seems to us important to gather together all the data available in the study of the problem.

At the University of Chicago over a 17 year period (1927 to 1944), gastric cancer was diagnosed in 576 patients. During the first few years of the institution's existence, the admissions were few; during the last 10 years, they averaged 40 to 50 cases per year.

Sex and age incidence. Chart 1 shows the striking relationship between gastric cancer and age, 76.3 per cent of the patients being over 50 years of age. This figure is almost identical with that found by Faged and Larsen, 77 per cent of 407 patients.

In order to see if a trend toward greater age could be demonstrated in our patients they were arbitrarily divided into two groups, (1) those from 1927 to 1938 (302), and (2) those from 1939 to 1944 (274). This trend was indeed evident for the patients over 50 constituted 73.7 per cent of the total in group 1 and 79.3 per cent of the total in group 2.

Sixty-nine per cent of the group were male and 31 per cent female. The ratio, though unexplained, is in accord with that generally reported by others.

Duration of symptoms. In 559 patients available for study, the duration of symptoms was less than 3 months in 26 per cent and less than 6 months in an additional 21.2 per cent. Thus in 47 per cent of the patients a diagnosis of carcinoma was made within 6 months or less after the onset of symptoms. Walters, Gray, and Priestley report that 18 per cent of their patients had symptoms of 3 months' duration and 37 per cent of less than 6 months. The higher percentage in our series may indicate a trend toward earlier diagnosis. Faged and Larsen in 375 similar cases seen over approximately the same period (1927 to 1942) found 40 per cent with symptoms of less than 3 months and 24 per cent of between 3 and 6 months' duration, a total of 64 per cent with symptoms of less than 6 months' duration. The suggestion is that the diagnosis of gastric cancer is made earlier in Denmark than it is in this country. Fifty per cent of the group (375), however, were considered inoperable at the time of the first examination.

Our study further showed that 24.5 per cent of group 1 (1927 to 1938) and 28.9 per cent of group 2 (1939 to 1944) had symptoms of less than 3 months' duration. Similarly 18 per cent of group 1 had complaints of 3 to 6 months' duration as compared with 24.8 per cent in group 2. These figures again suggest a trend toward earlier diagnosis. The importance of a roentgen-ray examination in every patient over 35 years who complains of gastrointestinal symptoms for even a few weeks is self-evident.

Palpable tumor mass. In keeping with the shorter duration of symptoms in the later group is the finding that 41.9 per cent of group 1 had palpable tumor masses in contrast to 33.8 per cent of group 2.

Free acid values. Gastric analysis, carried out in 266 patients (46.2%) disclosed a free acid value of over 25 units (histamine) in 32.4 per cent; in some, the titration reached 75 to 100 clinical units.

Gastroscopy. Gastroscopy has been in use in this institution since 1934. During this period, 481 patients with gastric cancer were

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seen; 163 (33.8%) were subjected to gastroscopy. In some, the obvious diagnosis and the poor physical condition made the examinations unnecessary. It is interesting to note that the correct diagnosis was made in 84.6 per cent of the patients so examined. In 15.4 per cent the examination was inconclusive due to the technical difficulties preventing satisfactory passage of the instrument or failure to visualize the lesion or the diagnosis was incorrect because of inability to differentiate neoplasm from ulcer, hypertrophic gastritis, lymphoma, and sarcoma. Schindler and Letendre reported that 89 per cent of 91 carcinomas were diagnosed correctly while the figure obtained by Halmos (cited by Baron) was 76 per cent.

The importance of prolonged and repeated gastroscopic examinations cannot be too strongly stressed as parts of the stomach not clearly visualized at one examination were frequently seen at the time of another examination several days or weeks later. The change in the appearance of a lesion was often so marked in a few days that the examiner was then able to make a correct diagnosis even though this was not possible at the time of initial examination. Differences in peristaltic action, spasm, and the superficial appearances of a lesion may well account for the changing picture. While gastroscopy is a valuable adjunct in the diagnosis of gastric carcinoma the criteria for determining the resectability of a lesion by this method need further study.

X-ray examination. X-ray examination is the most important single diagnostic procedure. In the group of 576 patients, 92.8 per cent received roentgen examination in our institution; some of the remainder had been examined elsewhere; in others, the advanced lesions and evident metastases made the procedure unnecessary and even undesirable. Of 533 patients subjected to x-ray examination, a definite correct diagnosis was made in 92 per cent. In 8 per cent, the differential diagnosis of benign and malignant ulceration or benign and malignant pyloric obstruction was very difficult and the results not conclusive. Repetition of the examination in difficult cases was of great value.

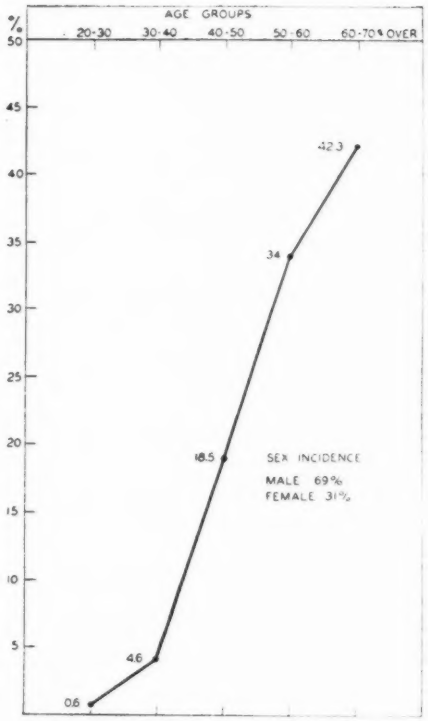

Chart 1. Age and sex incidence in 566 cases.

It is worth while to note that the x-ray examination is not entirely reliable in determining the resectability of a lesion. In 400 cases, at the Mayo Clinic (5), about 50 per cent of those thought to be roentgenologically suitable for resection were found unsuitable at operation and on the other hand 39 others deemed unresectable on x-ray examination were removed at operation.

DISCUSSION

These figures seem to us to substantiate the recognized high incidence of gastric cancer and to indicate a steady increase with the advancing general age level. The ideal of prompt and early diagnosis can only be achieved if all patients of "cancer age" with "dyspeptic symptoms" are submitted at once to objective study by means of the x-ray and the gastroscope. Even if this is done, it is probable that true "early" diagnosis will be

realized in only a relatively small per cent of the patients so afflicted, for the disease is, in its earliest phases, notoriously asymptomatic. For this reason St. John, Swenson, and Harvey examined the feasibility of routine periodic gastric x-ray examinations. Three unsuspected neoplasms were found in 2,413 asymptomatic men and women over 50 years of age. The cost of the procedure was low, but nevertheless such mass examinations are probably not practical. Furthermore, the number of lives actually to be saved by "early diagnosis" and "early operation" is conjectural, for our knowledge of the natural history of the various types of gastric cancer is very incomplete. Nevertheless "early diagnosis" and "early operation" do now constitute the only real hope for the victim of gastric cancer.

SUMMARY AND CONCLUSIONS

1. The incidence of gastric cancer will continue to rise as the average age of the population rises.

2. The incidence of gastric cancer rises sharply with advancing age, approximately three-fourths of the cases occurring in people over 50 years.

3. A trend toward earlier diagnosis is detectable.

4. The symptoms at the time the diagnosis was made were of less than 3 months' duration in one-fourth of our cases, of less than 6 months' duration in 53.7 per cent.

5. Gastroscopy yielded the correct diagnosis in 84.6 per cent of the cases in which it was utilized.

6. X-ray examination provided the correct diagnosis in 91.8 per cent of the cases so examined.

7. "Dyspeptic" symptoms appearing in patients of the "cancer age" warrant prompt roentgenologic and gastroscopic study.

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CONTINUITY OF THE FASCIAE LINING THE ABDOMEN, PELVIS, AND SPERMATIC CORD

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MANY of the anatomical and clinical textbooks describe the subcutaneous fasciae of the abdomen, pelvis, and scrotum as forming continuous layers and indicate the clinical importance of this continuity; for example, in directing and limiting the spread of urine extravasated from the penile urethra. Yet some of these texts describe the fasciae lining the abdomen, pelvis, and spermatic cord as distinct and separate anatomical entities, each with slight clinical significance. Studies of adult specimens in the anatomical laboratories, operating room, and at autopsy, however, indicate that the fasciae within the abdomen, pelvis, and spermatic cord also are continuous layers and that this continuity may have practical clinical importance. Since the abdomen and pelvis develop as continuous parts of the body, and the components of the spermatic cord were originally parts of the abdomen and pelvis, the fasciae lining these regions in the adult should not be considered as separate entities, but as parts of continuous layers.

The literature on the internal fasciae of these regions is so abundant that a review of the publications, with many of the controversial points, will not be attempted in this paper. It is sufficient to state that the fascial layers lining the abdomen, pelvis, and spermatic cord have been described so differently and so many names have been applied to their parts by various authors, that the clinician as well as the student and teacher of gross anatomy have difficulty in understanding the many described subdivisions of these fasciae individually, as well as the relation and continuity of these subdivisions with each other.

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A stratification of the abdominal fasciae into three layers in the adult was suggested by Daseler and Anson (1943): an internal layer for the digestive tube with its vessels and nerves; an intermediate layer for the kidneys, adrenals, ureters and their associated vessels and nerves, together with the aorta and vena cava; and an external layer for the parietal musculature (body wall) with its nerve and blood supply. This simplified concept of the stratification of the abdominal fascia was verified by Tobin (1944) in studies concerning the embryological origin and adult structure of the renal fascia. The question then arises, are the fasciae of the pelvis and spermatic cord divided into similar strata and are the fascial strata of the abdomen, pelvis, and spermatic cord continuous. This simplified working concept would help to clarify much of the confusion relating to these fasciae.

Such a continuity has been suggested in a few publications. In Hinman's *The Principles and Practice of Urology* (1937, p. 247, Fig. 119[1]), the continuation of the perirenal fascia down over the ureters behind the bladder to the fascia covering the seminal vesicles and prostate is shown. Zieman (1942) has also described the "transversalis fascia" as forming a continuous sheet within the abdomen and pelvis.

The whole concept of the abdominal and pelvic fasciae (and ligaments), together with the lining of the spermatic cord, could be simplified and made more comprehensible if these fasciae were restudied in view of: (1) their origin and continuity during development; (2) their form and continuity in the adult; (3) their possible function as a barrier to, or pathway for, the spread of infection or of extravasated blood or urine; (4) the pos-

[1]The dissection shown in this picture was done by Dr. Louis Yglesias in Dr. R. M. Nesbit's laboratory at the University of Michigan Medical School (R. M. Nesbit, personal communication).

sible relation of the abdominal fascia to ureteric obstruction; and (5) their use as cleavage planes in the surgical approach to the organs in these regions.

MATERIAL AND METHODS

The early development of the connective tissue which forms the subsequent fasciae lining the abdomen, pelvis, and spermatic cord, was studied in slides of sectioned human embryos of various ages. The further development and more mature form of the fasciae were observed in dissected 8 and 9 month fetuses. The adult structure, relation and course of the fasciae were ascertained by progressive dissection of the layers of the abdomen, pelvis, and spermatic cord in 10 middle aged cadavers cut in various planes as well as observations on the course of these layers during dissection of adult cadavers in the gross anatomy laboratory. Histological preparations of material obtained at biopsy or from autopsied bodies were also used to study the structure and relations of the fascial layers in unembalmed material. The histological preparations both from the embryonic and adult tissues were stained with Mallory's connective tissue stain to aid in studying the tissues forming the fasciae.

DEFINITIONS

Since so many different names and definitions have been given for the fasciae and other tissues to be described in this paper, the following definitions of the terms to be used are suggested as an aid in understanding these structures individually and in their relation to one another.

Peritoneum. The definition of this term by histologists and gross anatomists has a wide range of interpretation depending on the layers of tissue to be included. The concept suggested here is that generally used by the gross anatomist and surgeon. Therefore, the term *peritoneum* hereinafter is used to include the mesothelial cells, basement membrane and that part of the subjacent connective tissue, containing the terminal vessels and nerves of the peritoneum, which can be dissected readily as a layer from the connective tissue of the viscera or body wall (Fig. 1).

Retroperitoneal tissue. The tissue between the peritoneum and the intrinsic fascia of the viscera and the body wall is areolar, fibrous, or fatty in consistency, varying in its composition with the area under consideration and the body type of the individual. Like the peritoneum, it is a continuous layer and forms an embedding matrix for the nerves, vessels, and viscera of the abdomen and pelvis. It may be protecting or supporting tissue, either by its fatty nature or by the formation of ligamentous supports—some of which may contain bundles of smooth muscle tissue. This tissue has been described under many names: tela subserosa, subperitoneal, extraperitoneal, etc., tissue.

Transversalis fascia. This fascia is usually referred to in anatomy and surgery but, from the many contradictory definitions given in the anatomical and surgical texts and in the medical dictionaries, a clearer definition of this term appears to be needed. This fascia has been defined as: (1) all the tissue between the transversus abdominis muscle and the peritoneum; (2) the tissue between the investing fascia on the inner surface of the transversus abdominis muscle and the retroperitoneal tissue; and (3) the investing fascia on the inner surface of the transversus abdominis muscle.

The first definition of this fascia is the one given in most anatomical and surgical texts. It apparently originated from, and is perpetuated through, an erroneous interpretation of Sir Astley Cooper's (Fig. 2) original description of this fascia. His first work on hernia (1804) described and illustrated (Fig. 3), but did not name, this fascia. The term "transversalis fascia" was used for the first time in Part II of his work (1807, p. 3 and p. 4) where it was described as:

"Anteriorly to the crural arch (Poupart's ligament) is situated another fascia, which may be divided into two portions, the one being placed a little before the other. Its outer portion is attached to the greater part of the posterior edge of the crural arch, from as far as the spinous process of the ilium, and the inner labium of that bone, and it ascends to the posterior part of the transversalis muscle on the outer side of the spermatic cord, where that cord first quits the abdomen: the inner portion is thinner than the former, but is strengthened in this forepart by the tendon of the transversalis muscle; it is also slightly

attached to the crural arch, but is more firmly fixed to the ligament of the pubis, and is extended over the pubis into the cavity of the pelvis; it rises to the tendon of the transversalis muscle on the inner side of the spermatic cord, and is firmly attached to the linea semilunaris.

"This anterior fascia, which is described in the first part of this work (1804), should be called *fascia transversalis;* it lines the posterior part of that muscle, and there prevents the viscera from descending between the fibers of the abdominal muscles, but leaves an opening for the passage of the spermatic cord and round ligament, which I have ventured to name the *internal abdominal ring.*"

The following statement from the later editions of Cooper's work (edited by C. Aston Key, 1827 and 1844) apparently is the source of the widely used, but erroneous, definition—that the fascia transversalis is composed of all the tissue between the transversus abdominis muscle and the peritoneum (1844 edition, p. 26 and p. 27):

"When the lower portions of the internal oblique and transversalis muscles are raised from their subjacent attachment, a layer of fascia is found to be interposed between them and the peritoneum, through which the spermatic vessels emerge from the abdomen. This fascia, which I have ventured to name *fascia transversalis*, varies in density, being strong and unyielding towards the ilium, but weak and more cellular towards the pubes."

A more thorough study of the text, and particularly the plates illustrating Cooper's works, indicate that his concept of the peritoneum included all the tissue between the mesothelial surface of the peritoneal cavity and the intrinsic fascia of the body wall. Therefore when the peritoneum was removed, the only tissue remaining between the inner surface of the transversus abdominis muscle and his concept of the "peritoneum," was the intrinsic fascia on the inner surface of the abdominal wall. This concept is shown in Figures 3 (male) and 4 (female), reproduced from Cooper's original works.[1] The vessels shown in these figures are denuded of their connective tissue coverings and the inner surface of the transversus and rectus abdominis muscles are exposed after the "peritoneum" is removed. If this concept of the peritoneum is to be maintained, then the aorta, vena cava, adrenals, kidneys, spermatic vessels, etc. are also contained in the "peritoneum"; for the connective tissue embedding the pelvic vessels and that adjacent to the intrinsic fascia of the body wall is part of a continuous layer which also embeds the abdominal vessels and viscera.[2] More recent studies designate the tissue embedding the vessels and viscera of the abdomen and pelvis, retroperitoneal tissue, extraperitoneal, subserosal, etc. which apparently Cooper interpreted as part of the peritoneum.

Subsequent classical papers on the anatomy and surgery of inguinal hernia by Scarpa, 1809, Bassini, 1890, and Halsted, 1893, generally conform to Cooper's original definition of the transversalis fascia.

The second definition of the transversalis fascia, given in several anatomical texts—the tissue between the investing fascia on the inner surface of the transversus abdominis muscles and the retroperitoneal tissue—could not be verified by studies in normal adult specimens. The tissue defined as the transversalis fascia in this concept is the outer part of the retroperitoneal tissue and it could not be dissected as a separate layer in many instances. The retroperitoneal tissue may be in the form of two laminae adjacent to the lower abdominal wall in some individuals, just as the subcutaneous fascia in this region is in the form of two laminae; but if this outer layer, termed the transversalis fascia by this definition, is followed cephalad and caudad, it is found to be a part of the retroperitoneal tissue. Its areolar and fatty nature also would not justify its being the transversalis fascia.

The third definition—the investing fascia on the inner surface of the transversus abdominis muscle (apparently the concept Cooper meant to convey in his first works, 1804–1807)—is becoming the accepted meaning of the transversalis fascia, due largely to the studies of Anson and his co-workers. The following quotation from a paper by Anson and McVay (1938, p. 189 and p. 190) aptly describes the relation of the transversus abdominis muscle to the transversalis fascia:

[1]Similar figures are reproduced in the 1844 edition of Cooper's *The Anatomy and Surgical Treatment of Abdominal Hernia* edited by C. Aston Key and published by Lea and Blanchard, Philadelphia.

[2]Cooper's concept of the peritoneum is further verified by the statement from p. 30 (1844): "Within the cavity of the abdomen the spermatic vessels receive no other covering but that which is derived from the peritoneum."

"The medially placed tendinous plate is, in almost all descriptions, correctly termed aponeurosis; but curiously enough, the inferior continuation of the same structure is called a fascia, or specifically, the "transversalis fascia." That the two are merely areas of the same layer is clearly evident in those cases—numerous in the laboratory, rare in books and journals—in which the muscle fibers terminate at a high level, leaving a broad expanse of the stratum entirely without fascicles. . . . The inferior portion, appearing then as the hinder wall of the small lunular interval between the inferior border of the musculature and the inguinal ligament, is described and labelled as 'transversalis fascia' a term which is applicable only to the internal layer of investing fascia of the transversus abdominis muscle. . . . it is impossible to make a clear separation between the transversalis fascia and the aponeurosis of the transtransversus muscle."

The term transversalis fascia used hereinafter conforms with the above description—the intrinsic, investing fascia on the inner surface of the abdominal wall. This fascia is not limited in its extent to the inner surface of the transversus abdominis muscle but is continuous with the intrinsic fascia of the other components of the body wall: iliac fascia, fascia of the pelvic diaphragm, fascia of the femoral sheath and canal, etc. Its relation to the other components of the ventral abdominal wall is shown in Figure 5, modified from a drawing published by McVay and Anson (1940).

Apparently many of the descriptions of the transversus abdominis muscle and the transversalis fascia did not take into consideration the fact that anatomically and physiologically "a muscle" consists of both the aggregate of contractile elements and their binding and investing connective tissue. It appears from some descriptions of the transversalis fascia that the authors removed the investing fascia from the inner surface of the muscular part of the transversus abdominis muscle and yet considered its lower part, which does not contain muscular fibers in many individuals, the transversalis fascia. The error created by such a concept has resulted in confusing descriptions regarding the continuation and representation of the transversalis fascia in other parts of the abdomen or pelvis.

The same anatomical and physiological concept of a muscle is used for the subsequent discussion of the cremaster muscle and cremasteric fascia in their relation to the internal spermatic fascia, and for the formation of the internal spermatic fascia from the transversalis fascia.

Internal spermatic fascia. Exclusive of the skin and dartos fascia, three fascial layers are usually described as covering the vessels, nerves, lymphatics, vas deferens, testis, and tunica vaginalis: the external spermatic (intercrural or intercolumnar) fascia derived from the aponeurosis and fascia of the external oblique muscle; the cremasteric layer of muscle and fascia derived from the internal oblique, and in some individuals also from the transversus abdominis; and the internal spermatic (infundibuliform, *tunica vaginalis communis*) from the transversalis fascia (Hinman, 1937; Callander, 1939; Grant, 1940; Gray, 1942; Lowsley and associates, 1942; McVay and Anson, 1942; Cunningham, 1943; Mainland, 1945; and others).

The described origin and form of the external spermatic fascia are easily verified. The cremasteric muscle and fascia, however, have been described as arising either mainly from the internal oblique and only partly from the transversus abdominis muscle (Piersol, 1930; Howell, 1939; Gray, 1942; Morris, 1942) or equally from both muscles (Spalteholz, 1937; Grant, 1940), as is regularly the case in most other primates (Howell, 1939). The cremasteric muscle was observed to arise from both the internal oblique and the transversus abdominis muscles in those individuals whose transversus abdominis muscle had contractile fibers as far caudad as the inguinal ligament. Consequently the cremasteric fascia should be derived from and continuous with the fascia of these muscles. If the intrinsic fascia of the transversus abdominis muscle and the transversalis fascia are the same and therefore a source of the cremasteric fascia, then the internal spermatic fascia must be derived from, and continuous with, some tissue other than the transversalis fascia.

Although the internal spermatic fascia is illustrated in all the standard works on the spermatic cord, and a definite fascial layer can be found internal to the cremasteric layer in cases where hernia of long duration has resulted in thickening of all the layers of the spermatic cord, such a fascia is difficult to

demonstrate in dissections of normal spermatic cords. It is interesting to note that Cooper, 1804 (p. 8), did not describe the presence of a fascial layer between the cremasteric layer and the hernial sac:

> "When an inguinal hernia is dissected, immediately under the skin of the scrotum is found a fascia of greater or less thickness according to the duration and size of the tumor. This fascia is given off by the tendon of the external oblique muscle above the inguinal ring. . . . Under this fascia is the cremasteric muscle, which forms another covering to the hernial sac. . . . When this fascia and the cremaster muscle are removed, the proper hernial sac becomes exposed to view."

Histological sections of normal spermatic cords illustrate the intimate blending of the connective tissue around the cremasteric muscle with the inner fat and areolar tissue, forming the embedding matrix for the constituents of the spermatic cord (Fig. 6). Thorek (1924) and Piersol (1930) show similar pictures of sections through the spermatic cord.

In addition to the three customarily described (external and internal spermatic and cremasteric) layers, several authors (Deaver, 1927; Galludet, 1931; Singer, 1935; Jennings, Anson, and Wright, 1942; and Mainland, 1945) describe an additional inner layer of areolar and adipose tissue, derived from the retroperitoneal tissue, which forms the matrix around the vessels, nerves, lymphatics, vas deferens, etc. of the spermatic cord. The present study confirms the presence of this inner layer, which may contain a considerable amount of fat in some individuals and be the site of formation of lipoma (Cecil, 1927) or even be mistaken for herniation of retroperitoneal fat. This areolar and fatty tissue apparently is a normal constituent of the spermatic cord. It is so fused with the inner surface of the cremasteric fascia that the two cannot be separated easily by dissection in individuals without indirect inguinal hernia.

Therefore the term internal spermatic fascia is used to include all the connective tissue and fat which form the embedding matrix for the components of the spermatic cord internal to the fascia ensheathing the cremasteric muscle. This tissue is continuous with the retroperitoneal tissue of the abdomen and pelvis.[1] The older term, tunica vaginalis communis,[2] seems to be more appropriate than the term internal spermatic fascia, for this is not a definite fascial layer such as the external spermatic and cremasteric layers, but a common, embedding layer of areolar and fatty tissue for all the components of the spermatic cord.

ANATOMICAL OBSERVATIONS

Embryos. The continuity of the fasciae lining the abdomen, pelvis, and spermatic cord, of the adult, and the analogy of these fasciae with the subcutaneous fasciae, can be more easily understood by tracing briefly the development of these fasciae in their relation to the developing body wall and viscera.

In young embryos, a continuous sheet of mesenchyme (mesoblastic cells) fills all the potential space between the epithelium of the developing skin, on the outer surface of the body, and the mesothelium of the developing peritoneal (celomic) cavity, on the inner surface of the body not occupied by the vessels and viscera (Fig. 7).

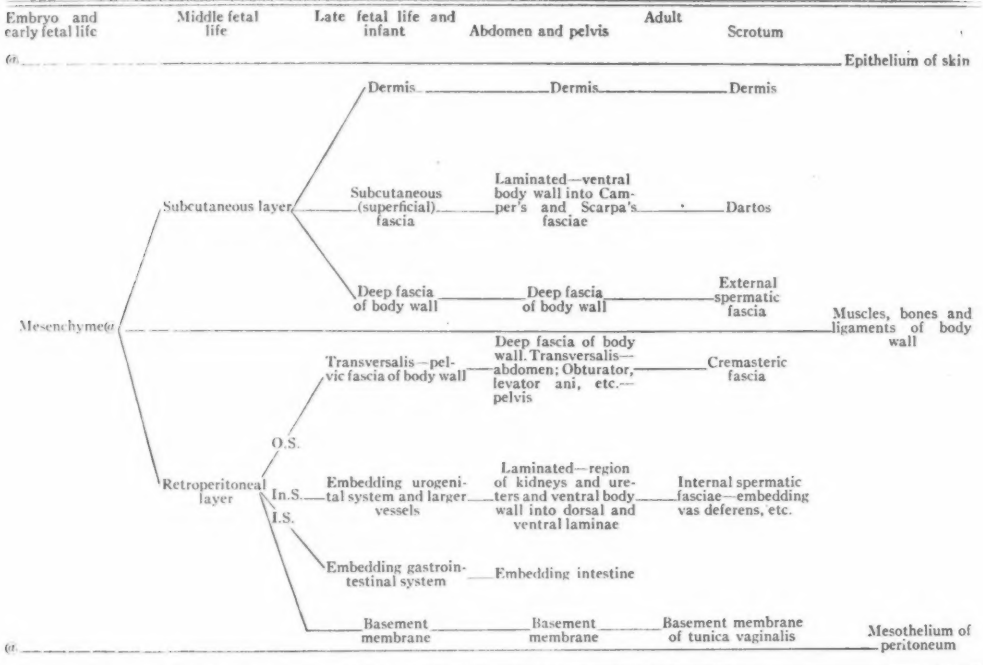
With the formation of the musculobony tissue in the body wall, the original mesenchymal sheet is divided into an outer, subcutaneous layer, associated with the skin and external surface of the musculobony tissue, and an inner, retroperitoneal layer, between the mesothelium of the peritoneal cavity and the inner surface of the musculobony tissue (Fig. 8). These layers are adjacent to the surface of the developing cutaneous and peritoneal epithelia, and form an embedding matrix for the myogenic and osteogenic cells of the body wall and the myogenic cells of the visceral wall—in which the muscles and bones develop and become ensheathed.

Emphasis will be placed mainly on the retroperitoneal tissue. This tissue embeds the digestive and urogenital systems together with their nerves and blood vessels in a continuous

[1] In editing Cooper's work, Key (1844) apparently noted the continuity of the abdominal fascia into the spermatic cord, for he wrote in the footnote to p. 30: "A process from the inner margin of the internal ring may be traced upon the spermatic artery and vein for some distance in the abdomen, resembling the thin fascia sent by the outer margin along the cord within the canal."

[2] The cremasteric fascia, cremasteric muscle and *tunica vaginalis communis* are the only coverings of the spermatic cord listed in the Basle *Anatomical Nomenclature* (Emmel, 1927).

TABLE I.



(broken lines)—structures before definitive form of tissue is developed.
O.S., In. S., I.S.—outer, intermediate, and inner stratum of retroperitoneal tissue.

layer of connective tissue, and will form the fascial lining of the abdomen and pelvis.

The connective tissue (fascia) of the ventrally placed digestive system and that of the laterally placed urogenital system, in the abdomen, are not difficult to understand because of the relatively constant, spacial relationship of these systems to each other and the mediolateral continuity of their embedding fascia (Fig. 8). With the change in position to a dorsally placed intestine and ventrolaterally placed urogenital organs, a misleading spacial change in fascial continuity arises which can be better understood by a brief review of the development of the pelvic organs.

In embryos before the 7th week, the terminal ends of the digestive and urogenital systems empty into a common structure, the cloaca, which is embedded in a layer of pelvic connective tissue continuous with that around the same systems in the abdomen. As the cloaca is divided into its two parts—the future hind gut and the urogenital sinus—the peritoneal (celomic) cavity intervenes between these two structures, separating the posterior surface of the developing bladder, prostate, and seminal vesicles from the anterior surface of the future rectum. This intervening peritoneal cavity is lined by a surface layer of mesothelium and subjacent retroperitoneal tissue. Therefore, although the retroperitoneal tissue around the terminal ends of the urogenital and digestive systems appear to be completely separated in one area, it is continuous laterally with similar tissue common to all the structures of the abdomen and pelvis (Fig. 9). With increasing age the mesenchymatous retroperitoneal tissue differentiates into the muscles and connective tissue of the developing viscera, and the adipose and areolar tissue filling the spaces between the body wall and the viscera (Fig. 10).

As the viscera change their positions, grow larger, and become functionally distended, the cul-de-sac of peritoneum between the lower ends of the rectum and urogenital systems becomes obliterated by caudocraniad fusion and regression of the mesothelial layers. The retroperitoneal tissue remains to form the future Denonvilliers' fascia of the adult (Cunéo and Veau, 1899; Smith, 1908; Wesson, 1922 and 1923; Tobin and Benjamin, 1945).

A similarly misleading change also applies to the fascia of the spermatic cord, the constituents of which begin development within the retroperitoneal tissue of the abdomen and pelvis. As these constituents descend through the layers of the abdominal wall into the scrotum, derivatives of these layers are also carried along as the external spermatic and cremasteric fasciae and the cremaster muscle. The processus vaginalis, a part of the peritoneal cavity, extends almost directly through the abdominal wall, carrying along the adjacent retroperitoneal tissue (internal spermatic fascia) embedding the constituents of the spermatic cord, into the scrotum.

Fetus. For the sake of brevity, a detailed description of the fasciae of the 8 and 9 month fetus will not be presented as the general form and continuity of their fasciae are similar to those of the adult, discussed subsequently in this paper. The composition of the continuous fascial layers lining the abdomen, pelvis, and spermatic cord in these age groups is more areolar and less collagenous than in the adult. There is less fat in the pararenal and perirenal[1] regions and in the other parts within the body, where deposits of adipose tissue would normally be found in the adult. The areolar and fatty tissue lining the spermatic cord is continuous with the retroperitoneal tissue of the abdomen and pelvis.

Adult. The connective tissue between the peritoneum and the body wall forms a continuous lining for the abdomen, pelvis, and spermatic cord. This continuity can be demonstrated by dissecting the peritoneum from the body wall and viscera, in addition to exposing the spermatic cord by reflection of the entire length of cremasteric layer (Fig. 11). The dissectible strata of this connective tissue: (1) an inner stratum associated with the digestive system and its nerve and blood supply; (2) an intermediate stratum embedding the adrenals, urogenital system, their nerve and blood supply together with the aorta and vena cava; and (3) an outer stratum, the intrinsic fascia of the components of the body wall; will be discussed individually and in their relation to each other, since all three strata were originally derived from, and form parts of, continuous layers of retroperitoneal connective tissue (Table I).

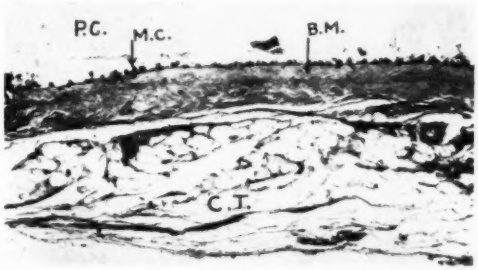


Fig. 1. Photomicrograph of a transverse section through an area of peritoneum dissected from the lateral wall of the abdomen of a body at autopsy. Blood cells are in the peritoneal cavity, *P.C.*, and adjacent to the mesothelial cells, *M.C.* The relation of the basement membrane, *B.M.*, to the subjacent connective tissue, *C.T.*, containing fat and terminal vessels and nerves, is shown. ×57.

Most of the peritoneum covering the viscera and lining the body wall is in the form of a single layer and therefore has only one subjacent layer of connective tissue associated with it. In some parts of the abdomen and pelvis, however, where rotation of the gut or obliteration of a peritoneal cul-de-sac has occurred, the connective tissue components of the several layers of embryonic peritoneum remain. Such a fusion of the embryonic peritoneal layers has been described by Toldt (1893) for the intestine; and Denonvilliers (1836 and 1837), Smith (1908), and Tobin and Benjamin (1945) for the pelvic cul-de-sac of peritoneum.

The inner stratum is mainly a thin areolar layer immediately outside of the entire extent of the peritoneal basement membrane. This inner stratum embeds and forms the adventitial layer of the abdominal and pelvic parts

[1]The terms perirenal and pararenal are used since they have become so well established in the literature. Etymologically, however, perinephric and paranephric are correct since both parts of these words are of Greek derivation; whereas perirenal and pararenal combine Greek with Latin derivations (Simeone, 1942).

Fig. 2. Photograph of a portrait of Sir Astley Paston Cooper (1768–1841). Reproduced from *Guy's Hospital Reports*, 1940–1941, 90: 73.

of the gastrointestinal system together with its vessels and nerves, in their course between the peritoneal layers of the mesenteries. It is continuous with the connective tissue capsules and stroma of liver, spleen, pancreas, etc.

This tissue can be demonstrated on any part of the intestine, but it is most easily seen grossly by cutting the peritoneum along the midaxillary line and reflecting it medialward. The connective tissue between the peritoneal layers remaining from the primitive mesenteries of the ascending or descending colon can be seen embedding the vessels and nerves to these structures (Fig. 11). The part of this layer for the gastrointestinal system begins on the esophagus at the diaphragm and, at this site, blends with the other strata of the abdominal connective tissue which are also attached to the ventral surface of the diaphragm. Caudally this stratum follows and covers all the contours of the gastrointestinal system together with its vessels and nerves, to fuse with the outer stratum on the pelvic diaphragm where the rectum is adjacent to the fascia of the levator ani muscles (Fig. 17).

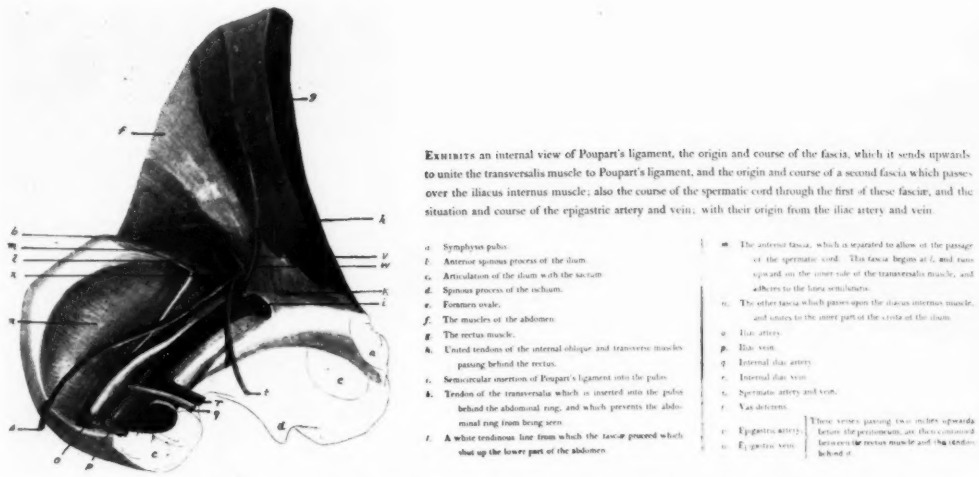
In other parts of the abdomen and pelvis not occupied by the gastrointestinal system this stratum is absent; except when inflammation results in thickening all the strata.

The intermediate stratum forms the embedding matrix for the adrenals, the urogenital system and the larger vessels and nerves (Figs. 11, 12, and 13). Its composition varies with the area under consideration—being areolar, fibrous, or fatty, depending on the mobility of the viscera in the area and the obesity (body type) of the individual. The fatty constituents of this stratum may be the site of lipoma formation (Ockuly and Douglass, 1937).

The representation of this stratum on the ventral abdominal wall is not illustrated in its entirety, but areas from this region are shown in Figure 5 to demonstrate its relation both to the peritoneum and to the transversalis fascia, and in Figure 16 showing a lamina of this tissue (umbilical vesical fascia) which embeds the urachus and lateral umbilical ligaments in a common layer, to become continuous, caudally, with the connective tissue of the bladder, seminal vesicles, and prostate. Hammond, Yglesias, and Davis (1941) have described the embryological origin, adult structure, and potential fascial space between the umbilical vesical fascia and the umbilical prevesical fascia. The latter apparently is the tissue anterior to the umbilical vesical fascia; which is part of the intermediate stratum, in those individuals where the intermediate stratum is in the form of two laminae adjacent to the ventral abdominal wall.

This intermediate stratum extends across the midline to enclose the aorta, vena cava, and renal vessels in a common layer (Fig. 13). Blood vessels for the gastrointestinal system or the muscular part of the body wall, are ensheathed in a continuation of this layer, thereby establishing a continuity of the intermediate with the inner and outer strata along the course of these vessels.

In the region of the kidneys and ureters this tissue may be in the form of two laminae, particularly in obese specimens. Local thickenings of the ventral lamina form the renal fascial layers of Gerota and the perirenal fat. The dorsal lamina forms the pararenal fat and areolar tissue between the ventral lamina and the transversalis fascia (Figs. 11, 12, 13). In



EXHIBITS an internal view of Poupart's ligament, the origin and course of the fascia, which it sends upwards to unite the transversalis muscle to Poupart's ligament, and the origin and course of a second fascia which passes over the iliacus internus muscle; also the course of the spermatic cord through the first of these fasciæ, and the situation and course of the epigastric artery and vein; with their origin from the iliac artery and vein.

a. Symphysis pubis.
b. Anterior spinous process of the ilium.
c. Articulation of the ilium with the sacrum.
d. Spinous process of the ischium.
e. Foramen ovale.
f. The muscles of the abdomen.
g. The rectus muscle.
h. United tendons of the internal oblique and transverse muscles passing behind the rectus.
i. Semicircular insertion of Poupart's ligament into the pubes.
k. Tendon of the transversalis which is inserted into the pubes behind the abdominal ring, and which prevents the abdominal ring from being seen.
l. A white tendinous line from which the fasciæ proceed which shut up the lower part of the abdomen.
m. The anterior fascia, which is separated to allow of the passage of the spermatic cord. This fascia begins at *l*, and runs upward on the inner side of the transversalis muscle, and adheres to the linea semilunaris.
n. The other fascia which passes upon the iliacus internus muscle, and unites to the inner part of the crista of the ilium.
o. Iliac artery.
p. Iliac vein.
q. Internal iliac artery.
r. Internal iliac vein.
s. Spermatic artery and vein.
t. Vas deferens.
u. Epigastric artery. } These vessels passing two inches upwards before the peritoneum are then contained
v. Epigastric vein. } between the rectus muscle and the tendon behind it.

Fig. 3. Photograph of Plate II and its legend from Cooper's first work on hernia (1804), showing the relation of the fascia, labelled *m*, (named the *transversalis fascia* in his 1807 work) to the vessels and components of the ventral body wall of the male. The "peritoneum" has been removed. (The letters and guide lines were so faded in the original plate, they were retouched in this photographic reproduction.)

specimens of slight obesity this tissue, caudal to the renal region, may not be laminated but consist only of a single stratum (Tobin, 1944).

Cephalad, the ventral lamina encloses the adrenals and kidneys in separate compartments and fuses with the dorsal lamina and outer stratum, both of which are also attached to the ventral surface of the diaphragm (Figs. 11, 12). The ventral lamina continues caudally around the spermatic vessels and ureters and fuses with the connective tissue of the bladder (Figs. 11, 14, 15, and 17). At the abdominal inguinal ring the ventral lamina, surrounding the spermatic vessels from kidney region, and part of the intermediate stratum around the vas deferens, from bladder region, fuse with the dorsal lamina and continue as the innermost layer, internal spermatic fascia, of spermatic cord (Figs. 11 and 12).

The autonomic nerves, around the aorta, are also embedded in this stratum. A division of these nerves, the hypogastric plexus, embedded in a derivative of this stratum, leave the iliac vessels at the level of the 2nd or 3rd sacral vertebra (Figs. 14 and 15) and continue their course in the intermediate stratum embedding the pelvic viscera. A similar course

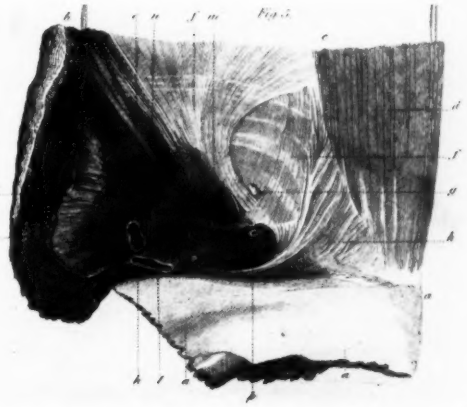


Fig. 4. Photograph of Figure 5 from Plate II of Cooper's (1807) work on hernia. This figure illustrates the relation of the transversalis fascia to the muscles of the abdominal wall and the vessels after the "peritoneum" had been removed from a female body. *a*, Pubis; *b*, ilium; *c*, abdominal muscles; *d*, rectus; *ff*, junction of fascia iliaca and transversalis behind crural arch; *g*, round ligament passing from abdomen through fascia transversalis; *h*, portion of fascia or tendon passing from pubis to join rectus; *i*, fascia iliaca; *k*, iliac artery; *l*, iliac vein; *m*, epigastric artery; *n*, epigastric vein; *o*, anterior crural nerve; *p*, crural space by which crural hernia descends: it leads into crural sheath.

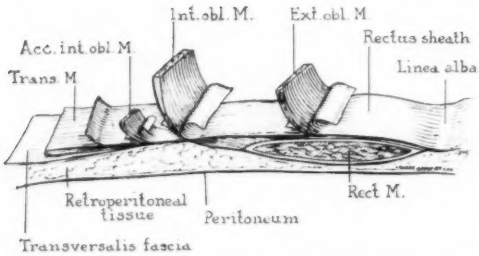


Fig. 5. Diagram of the relations of the peritoneum, retroperitoneal tissue, transversalis fascia, and abdominal muscles, with their fascial sheaths, after removal of the skin and subcutaneous fascia and partial dissection of the muscles. (Modified from McVay and Anson, 1940.)

of the pelvic autonomic nerves is described for the female by Curtis, Anson, Ashley, and Jones (18, 1942).

Denonvilliers' fascia, a dense collagenous and muscular layer, is located between the intermediate stratum on the posterior surface of the bladder, prostate, and seminal vesicles and the inner stratum on the anterior surface of the rectum. Denonvilliers' fascia also fuses laterally with the areolar and fatty tissue of the intermediate stratum (Fig. 17).

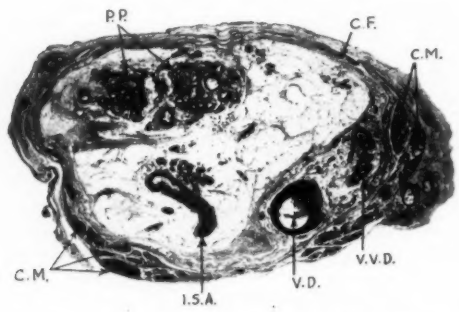


Fig. 6. Photomicrograph of a transverse section through a spermatic cord removed from an adult at operation. The skin, dartos, and external spermatic fascia were incised and the cremasteric muscle and fascia with the enclosed contents of the spermatic cord were fixed for sectioning. Note the cremaster muscle, *C.M.*, is embedded in a circular layer of cremasteric fascia, *C.F.* No definite fascial layer is found internal to the cremasteric fascia, which could be termed the internal spermatic fascia, other than the fatty and areolar tissue embedding the internal spermatic artery, *I.S.A.*, pampiniform plexus of veins, *P.P.*, vas deferens, *V.D.*, vessels of the vas deferens, *V.V.D.*, and nerves. Connective tissue strands encircle the vas deferens and its vessels as well as the pampiniform plexus of veins. ×3.

The intermediate stratum is continuous with the areolar and fatty tissue in the space of Retzius—between the dense fascial collar of the prostate and bladder and the outer stratum of the pelvic wall. Smooth muscle fibers may be found in this layer in some locations, as in the puboprostatic ligaments. This stratum fuses with the outer stratum at the points where the viscera are attached to, or vessels and nerves enter into, the muscles and bones of the body wall.

The outer stratum. The outer stratum is associated with the muscular and bony components of the body wall. It is usually designated in a limited region as the transversalis fascia. However, this layer is not limited entirely to the extent of the transversus abdominis muscle but it can be traced as a continuous layer from the fascia covering the inner surface of the transversus abdominis muscle to other components of the body wall: rectus abdominis, iliopsoas, obturator, levator ani, etc. (Figs. 5, 12, 13). It is a thickened supporting

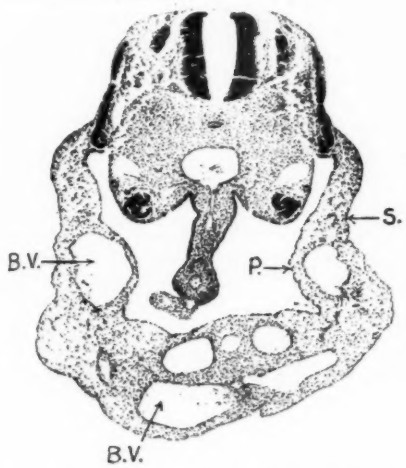


Fig. 7. Photomicrograph of a transverse section through a 6 millimeter crown-rump-length human embryo. The mesenchyme (connective tissue) between the developing skin, *S.*, and mesothelium of the developing peritoneal, *P.*, cavity is a continuous homogeneous layer in the areas not occupied by the embryonic blood vessels, *B.V.* The connective tissue around the developing intestine and within its mesentery is continuous with the connective tissue embedding the developing vessels and viscera in the dorsal part of the body. ×45.

layer fused to the inner surface of the various parts of the body wall.

Fascial collars appear to be formed from this outer stratum around the prostate and bladder and the lower part of the rectum, where they are attached to the fascia of the pelvic diaphragm (Fig. 17). Curtis, Anson, and Beaton (1940) have described fascial collars, derived from the pelvic fascia, ensheathing the female pelvic viscera. Connective tissue from the other strata is also connected to the pelvic viscera and is continuous with the cephalic part of the fascial collars—the connective tissue along the course of the ureters, vas deferens, blood vessels and nerves to the pelvic viscera, and on the sides of the peritoneal cul-de-sac and Denonvilliers' fascia fuse with the fascial collars of the prostate, seminal vesicles, bladder and rectum (Figs. 11, 14, 15, 16 and 17).

The outer stratum covers the ventral surfaces of the bodies of the vertebrae. It continues caudally to cover the ventral surface of the sacrum, and the vessels closely applied

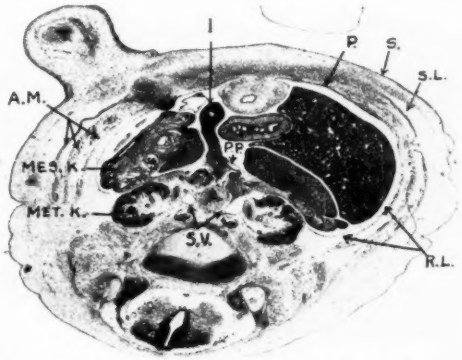


Fig. 8. Photomicrograph of a transverse section through a 19.5 millimeter human embryo. The developing abdominal muscles, *A.M.*, divide the mesenchyme between the developing skin, *S.*, and the peritoneum, *P.*, into a subcutaneous, *S.L.*, and a retroperitoneal, *R.L.*, layer. Note the continuation of the connective tissue in the mesentery of the intestine, *I.*, around the aorta, *A.*, subcardinal veins, *S.V.*, and the metanephric kidney, *MET. K.* The degenerating mesonephric kidney, *MES. K.*, and the parietal layer of peritoneum, *P.P.*, are indicated. ×15.

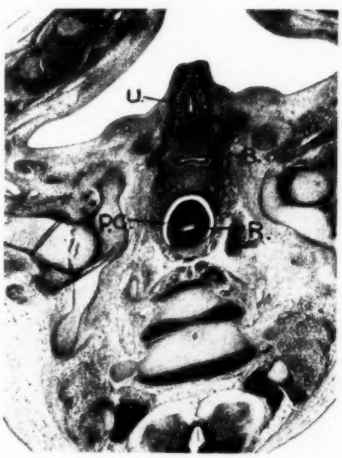


Fig. 9. Photomicrograph of a transverse section through the pelvis of a 19.5 millimeter human embryo. Connective tissue (mesenchyme) surrounds the developing rectum, *R.*, bladder, *B.*, and the developing muscles of the pelvic floor. This connective tissue is continuous with the layer around the digestive and urogenital systems in the abdomen. The peritoneal cavity, *P.C.*, surrounds three sides of the developing rectum and, in this area, separates the connective tissue around the rectum from that around the developing bladder. The position of the urethra, *U.*, is shown. ×10.

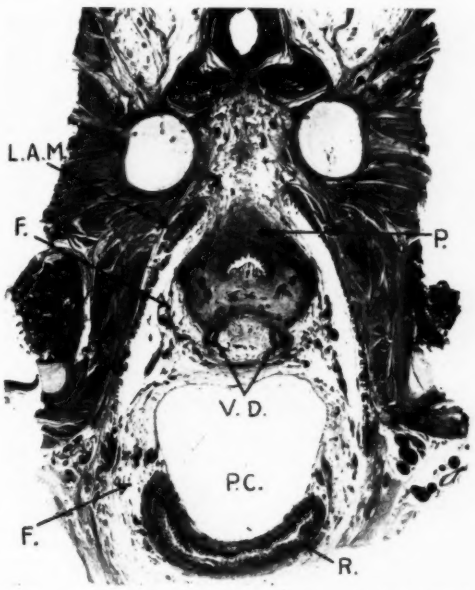


Fig. 10. Photomicrograph of a transverse section through the pelvis of a 208 millimeter, male human embryo. The muscles are forming around the pelvic viscera and in the pelvic floor. Mesenchyme has differentiated into: connective tissue around rectum, *R.*, vas deferens, *V.D.*, and prostate, *P.*; fat, *F.* in space of Retzius and on sides of rectum; and fascia adjacent to levator ani muscles, *L.A.M.* ×4.

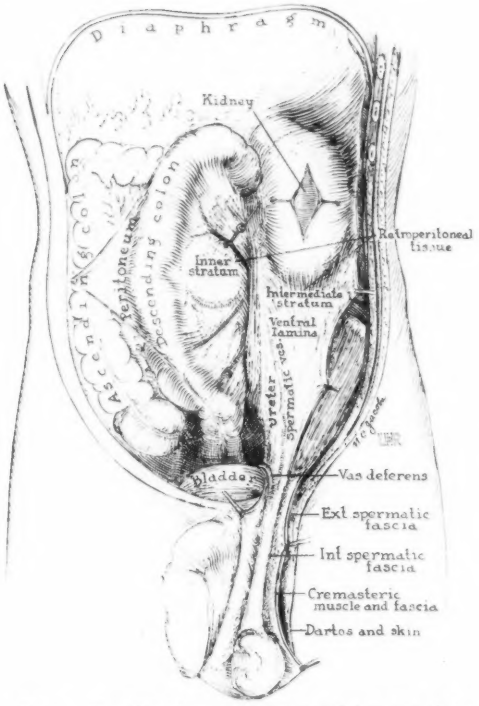


Fig. 11. Drawing of part of the fascia lining part of the abdomen, pelvis major, and spermatic cord. The ventral abdominal wall, liver, stomach, and small intestine have been removed and the extent of the internal spermatic fascia exposed by reflection of the skin, dartos, external and cremasteric layers. The parietal peritoneum was cut along the left midaxillary line, dissected from the subjacent retroperitoneal tissue, and reflected medialward. By this reflection, the relation of the peritoneum to the descending colon and its mesentery are shown. The colic vessels are embedded in the inner stratum of retroperitoneal tissue which also surrounds the colon. In one area part of the inner stratum has been removed from the colic vessels (elevated with hook). The ventral lamina of the intermediate stratum embeds the adrenals, kidneys, ureters, and spermatic vessels. Caudally the intermediate stratum surrounds the bladder and seminal vesicles and is continuous with the internal spermatic fascia around the components of the spermatic cord.

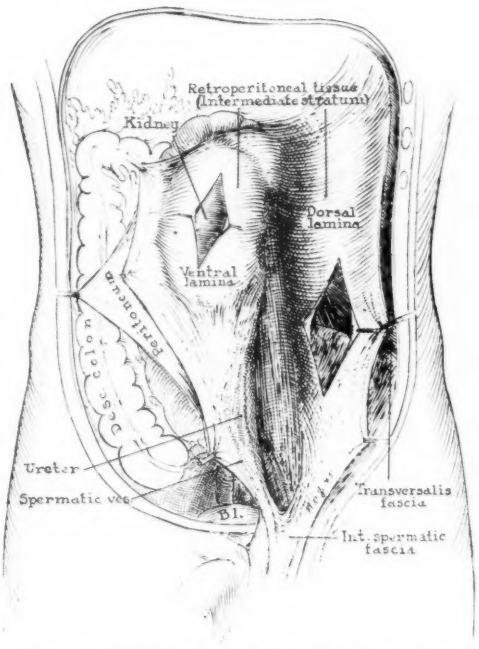


Fig. 12. Drawing from a further stage in the dissection shown in Figure 11. The intermediate stratum of retroperitoneal tissue consists of two laminae. The ventral lamina, embedding the adrenal, kidney, ureter, and spermatic vessels, is reflected medialward to expose the dorsal lamina of this stratum. Both laminae become continuous with the internal spermatic fascia at the abdominal inguinal ring. The dorsal lamina is shown in its relation to subjacent transversalis fascia.

to the surface of the sacrum and lateral pelvic wall. Although the craniad sources of these vessels were embedded in the intermediate stratum, in the pelvis the only dissectible fascial layer around these vessels was that continuous with the outer stratum (Fig. 15) usually designated as the pelvic fascia.

As branches of the pelvic vessels go to the viscera, sheaths of connective tissue are carried with them from the outer stratum to fuse with the intermediate stratum embedding the viscera. These connective tissue sheaths may be a partial support to the pelvic viscera as indicated by the case of the superior hemorrhoidal vessels and their ensheathing fascia supporting the upper part of the rectum. Curtis, Anson, Ashley, and Jones (1942) have suggested a similar rôle for the connective tissue around the blood vessels of the female pelvis.

CLINICAL CONSIDERATIONS

The structure and interconnections of the fascial strata lining the abdomen, pelvis, and spermatic cord limit or enhance their clinical applications. In regions of the body where there is little mobility between the parts, i.e.

on the ventral abdominal wall immediately above and lateral to the umbilicus, the fascial strata are so intimately blended, because of the paucity of intervening fatty and areolar tissue, that the peritoneum appears to be attached to the transversalis fascia. Such an arrangement does not permit surgical manipulation of the various strata and has no doubt led to the current misunderstanding of structure and continuity of these strata into the dorsal part of the abdomen and into the pelvis—since many of the original descriptions were made primarily by individuals interested in surgery of the ventral abdominal wall. However, where there is mobility between the viscera and the body wall, as in the renal region, in the pelvis, and in the scrotum, the number and thickness of the layers increase. It is in these regions that the fascial strata and the potential spaces between the strata have more clinical applications.

The anatomy of the fascial strata has been described in a preceding part of this paper. The potential fascial spaces between the strata

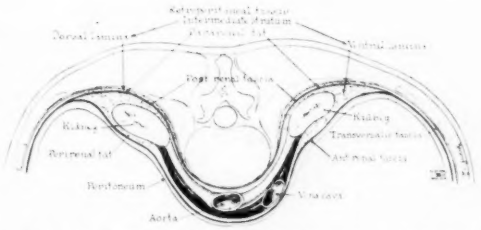


Fig. 13. Drawing of a cephalic view from a transverse section through the renal region. This section was dissected to show the derivatives of the intermediate stratum of retroperitoneal tissue. The dorsal lamina forms the pararenal fat between the transversalis fascia and posterior renal fascia. The ventral lamina forms the perirenal fat and the anterior and posterior layers of renal fascia. The retroperitoneal tissue is continuous across the midline, along the course of the renal vessels, from one kidney area to the other.

will be summarized and a few clinical examples given to indicate the importance of the strata or the potential spaces between the strata.

Potential fascial spaces of clinical importance are found:

1. Between the peritoneum and (a) the inner stratum around the gastrointestinal sys

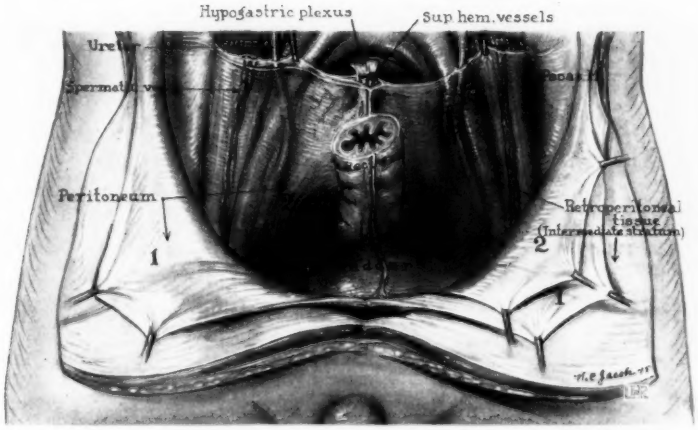


Fig. 14. Drawing of the relation of the peritoneum to the retroperitoneal tissue of the pelvis. On the left side of this figure, the parietal peritoneum of the abdominal wall, *1*, is freed from the subjacent intermediate stratum of retroperitoneal tissue, and the parietal and visceral pelvic peritoneum, *2*, are intact. On the right side the pelvic peritoneum, *2*, has been dissected from the intermediate stratum of retroperitoneal tissue and reflected ventrad. The spermatic vessels, ureters, superior hemorrhoidal vessels, and hypogastric plexus of nerves are embedded in the retroperitoneal tissue. The upper part of the psoas muscle, the iliac vessels, and sacrum are covered by the outer stratum of the retroperitoneal tissue which forms the fascia of the body wall. Note the potential space between the latter and the retroperitoneal tissue across the midline.

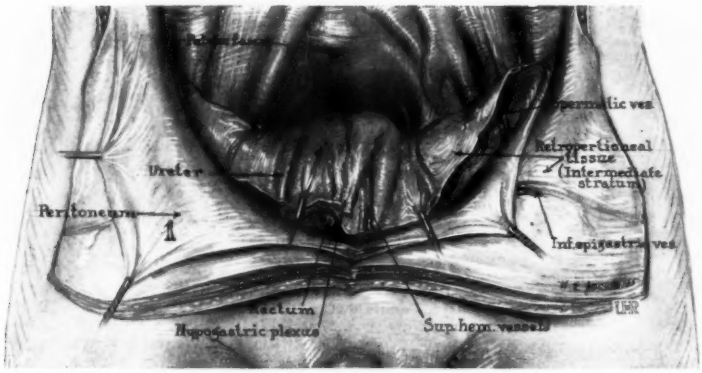


Fig. 15. Drawing of another stage in the dissection shown in Figure 14. The intermediate stratum of retroperitoneal tissue embedding the spermatic vessels, ureters, superior hemorrhoidal vessels, and hypogastric plexus is retracted forward together with the rectum embedded in the inner stratum. The continuation of the transversalis fascia as the pelvic fascia is shown. The inferior epigastric vessels are embedded in the intermediate stratum of retroperitoneal tissue on the ventral abdominal wall.

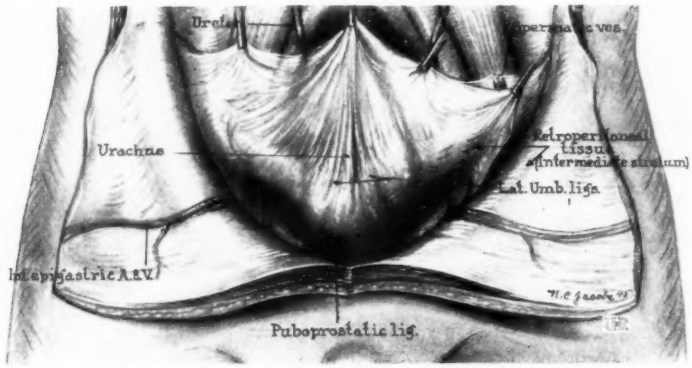


Fig. 16. Drawing from further stage in the dissection of the specimen shown in Figures 14 and 15, to illustrate the layer from the intermediate stratum of retroperitoneal tissue which embeds the urachus, lateral umbilical ligaments, and bladder. This layer is retracted from the intermediate stratum of the ventral abdominal wall. Some of the fat and areolar tissue in the space of Retzius, at the sides of the bladder, have been removed to show the continuity of the layers of the intermediate stratum with the fascia of the pelvic wall. The puboprostatic ligament is a local condensation of this fascia, containing bundles of smooth muscle, which attaches the prostate to the pubis. The course of the ureter, spermatic and inferior epigastric vessels is shown.

tem and within its mesenteries: or (b) the peritoneum and the intermediate stratum in the parts of the abdomen and pelvis not occupied by the gastrointestinal system.

2. Within the intermediate stratum; (a) where local thickenings of this stratum embed several structures in a common layer; (b) in regions where the intermediate stratum consists primarily of areolar tissue; and (c) where this stratum is laminated—the renal region and adjacent to the ventral abdominal wall.

3. Between the intermediate stratum and the outer stratum.

These are the anatomically dissectible spaces. The course of extravasated blood and urine from a traumatized vessel, kidney, o

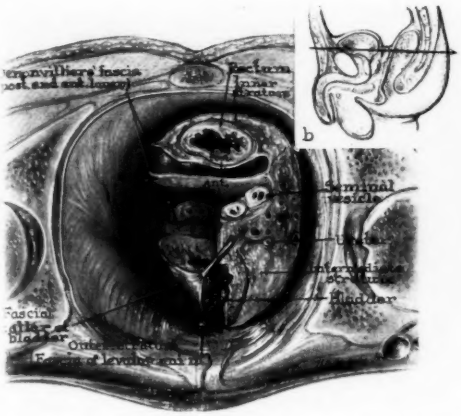


Fig. 17. Drawing, cephalic view, from a dissected, transverse section through the pelvis of the specimen shown in the preceding figure. This section was cut in the plane indicated by the insert, b. Part of the bladder and seminal vesicles have been removed. The fascia of the levator ani muscles (outer stratum) appears to be continuous with the fascial collar of the bladder. The ureter and the vesical vessels are embedded in the intermediate stratum of retroperitoneal tissue, which forms the fat and areolar tissue in the space of Retzius, at the sides of the bladder. The rectum, embedded in the inner stratum of retroperitoneal tissue, is shown in its relation to the posterior layer of Denonvilliers' fascia. The anterior layer of Denonvilliers' fascia, covering the posterior surface of the seminal vesicles and prostate is dissected from the posterior layer of Denonvilliers' fascia. Both of these layers fuse laterally with the intermediate stratum of retroperitoneal tissue. A potential space is found between the fascia around the rectum and the continuation of the outer stratum of fascia, over the posterior wall of the pelvis.

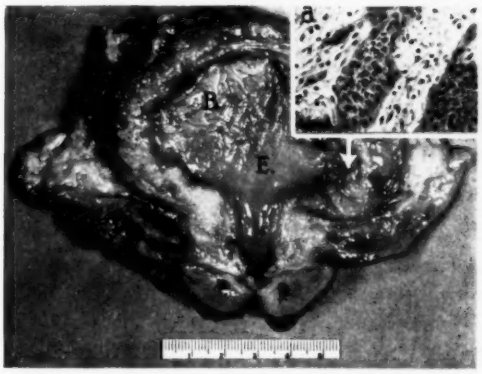


Fig. 18. Photograph of bladder and prostate removed surgically from B. A., 59 year old, white male, No. 180,302. The site of carcinoma in the bladder, *B*, is indicated by the arrow. The carcinoma cells are shown in the insert, a, a photomicrograph from a histological section of biopsied tissue. The area adjacent to the carcinoma was edematous, *E*. The prostate gland, *P*, together with its capsule, the seminal vesicles, and the bladder were removed intact. Dissection was made in front of the anterior layer of Denonvilliers' fascia (Fig. 17) to facilitate the removal of the seminal vesicles, prostate, and bladder, as a unit, and to prevent injury to the rectum and peritoneum. Insert ×120.

ureter, respectively, as well as infection from a kidney or perinephric abscess, may break through the various strata and involve one or more of these spaces.

Fascial strata as surgical guides. A knowledge of the fascial strata of the abdomen and pelvis is a great asset to the surgeon who operates in these regions. In approaching the kidney or the ureter through the lumbar incision, after the abdominal muscles and the transversalis fascia are incised, definite fascial planes are observed which can be followed along the dorsal posterior surface of the retroperitoneal tissue embedding the adrenals, kidneys, ureters, and gonadal vessels (Figs. 12 and 13).

In some cases of carcinoma of the bladder it becomes desirable to remove this organ together with the prostate gland, the seminal vesicles, and a portion of the urethra as a unit (Fig. 18). Whether one uses the procedure of Hinman (1935), Beer (1937) or others, the dissection and removal of this group of organs as a unit can be greatly facilitated by utilizing the potential space between the capsule of the prostate gland and seminal vesicles and the anterior layer of Denonvilliers' fascia; as previously pointed out by Wesson (1922 and 1923) and later suggested in the anatomical restudy of Denonvilliers' fascia by Tobin and Benjamin (1945).

In some cases where the ureter has been constricted by bands of retroperitoneal fibrous tissue, this structure becomes dilated in certain portions, narrowed in others, with considerable variation in the tortuosity. It is not possible to prove whether the fibrous bands cause, or result from, the tortuous ureters; because the kidney, ureter, and perirenal fascia develop together. Nevertheless Tobin (1944) reported absence of the perirenal fascia on the same side as the congenitally absent kidney and ureter. Apparently the presence of the kidney and ureter govern to some degree, at least, the thickening of the intermediate stra-

Fig. 19. Photograph of a pyelogram (skiodan) of S. B., 6 year old, white female, No. 150, 960. The right ureter is enlarged and tortuous. This patient had a unilateral trigone with absence of the left ureteral orifice, and presumably agenesis of the left kidney and ureter. The entire length of the right ureter was explored and the many dense bands of retroperitoneal tissue adherent to the ureter were severed. Compare with Figure 20.

Fig. 20. Photograph of a specimen obtained from autopsy of G. C., 3 week old, white male, No. 233,142. A congenital valve (indicated at the point of the arrow) was present in the prostatic urethra. The wall of the bladder, *B*, was hypertrophied and trabeculated. Thickened bands of retroperitoneal tissue were adherent to the dilated, tortuous ureters, *U*. The continuous layer of retroperitoneal tissue embedding the bladder, ureters, and spermatic vessels also forms the renal fascia around the kidneys, *K*. The relation of the aorta, *A*, to the other structures is indicated.

tum to form the fascia from the differentiating mesenchyme. It is possible to have a congenitally absent kidney and fascia on one side with normal development of these structures on the other side. On the other hand one can have a situation similar to the one aforementioned with marked changes in the ureter and fascia as shown in Figure 19, or the condition may be bilateral (Fig. 20). These malformations can be partly corrected by surgically severing the bands of retroperitoneal fibrous tissue and resecting the redundant portion of the ureter in the lower third and carrying out an oblique anastomosis (Hinman, 1929).

Perirenal insufflation. Many reports have been made of the spread of air injected around the kidney or adrenal for roentgenographic studies of pathology in these regions (Carelli and Sordelli, 1921; Rosenstein, 1921; Cahill, 1935; and others). The air may spread to the opposite renal space in about 10 per cent of the cases (Senger and Bottone, 1944), to the caudal surface of the diaphragm, or may follow the fascial planes along the ureter or gonadal vessels into the pelvis (Fig. 21). This spread of injected air can be readily understood, if reference is made to the potential spaces between the continuous fasciae around the kidneys, adrenals, together with the large blood vessels, and the ureters; and its continuation to the other side (Fig. 13), dorsally to

Fig. 21. Photograph of an x-ray film following perirenal insufflation of air in J. D., 18 year old, white female, No. 52,684. The air apparently entered the pararenal space on the right side (left side of figure) and continued caudally between the laminae of the intermediate stratum of retroperitoneal tissue toward the right inguinal canal. On the left side the air appears to have entered the perirenal space and does not continue as far caudally as on the right side. The fascial space between the adrenal, *Ad.*, and kidney, *K.*, is demonstrated by the injected air on the right side of the figure.

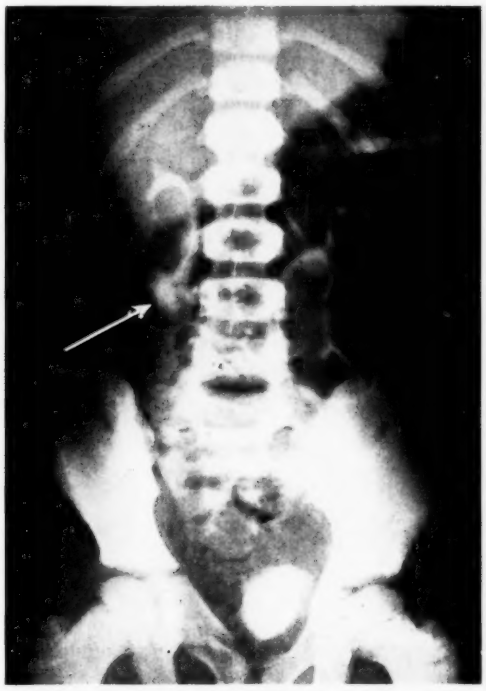


Fig. 22. Photograph of a 10 minute excretory urogram (diodrast) from W. S., 7 year old, white male, No. 209,305. Diodrast is extravasated into the retroperitoneal tissue as indicated by the arrow. This patient was hit by a passenger bus and at the time of surgery the right ureter was found to have been severed by an aberrant artery to the lower pole of the kidney. Diodrast, urine, and blood were found in the retroperitoneal tissue.

the diaphragm or ventrally into the pelvis (Figs. 11 and 15). To our knowledge no cases have been recorded where air has spread into the scrotum or labia major along the course of the spermatic cord or round ligament.

Retroperitoneal extravasation. Retroperitoneal extravasation of blood or urine from an injured kidney, ureter (Fig. 22), or rupture of the larger blood vessels, through trauma or by degenerative erosion from a pathological condition, is not uncommon. The course of such extravasation has been reported many times previously in both the male and female. The rôle of the fascial strata of the abdomen, pelvis, and spermatic cord in extravasation of blood or urine, for sake of brevity, will be illustrated therefore only by the following cases.

Nammack and Hirsch (1945) reported a case of rupture of the aorta, proximal to the bifurcation, with resultant retroperitoneal ecchymosis throughout the mesenteric-bowel junction, along the left side of the abdominal cavity, extending from the left perirenal region down the retroperitoneal tissue to the left scrotum. Externally there was considerable ecchymosis of the left inguinal region, the left lower quadrant, the upper portion of the left upper thigh and the left side of the scrotum.

The patient reported in this case had reducible bilateral inguinal hernias, which may account for the blood passing into the scrotum. After retrograde injection of liquid latex beneath the cremasteric layer of the spermatic cord toward the abdomen of unembalmed

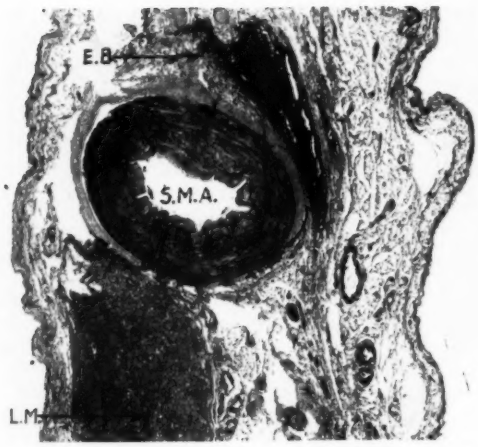


Fig. 23. Photomicrograph of a transverse section through the mesentery of the jejunum, obtained at autopsy, from H. P., 61 year old, white male, No. 52,684. This patient had both retroperitoneal and intraperitoneal hemorrhage following rupture of the left gastroepiploic artery. Extravasated blood, *E.B.*, is present in the retroperitoneal tissue of the mesentery near the superior mesenteric artery *S.M.A.* A mesenterial lymph node, *L.M.*, is shown. ×13.

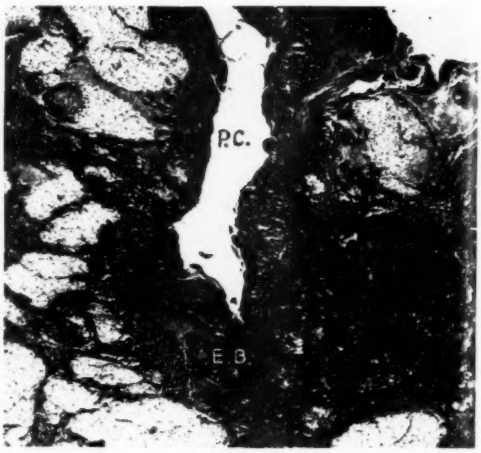


Fig. 24. Photomicrograph of a longitudinal section through the right inguinal sac obtained at autopsy from H. P., 61 year old white male, No. 52,684. Extravasated blood, *E.B.*, was found at the distal end of the herniated peritoneal sac located in the upper part of the right spermatic cord. The connective tissue adjacent to the mesothelium lining the peritoneal cavity, *P.C.*, was thickened. ×13.

bodies, we found the latex traversed the connective tissue within the spermatic cord only as far as the external abdominal ring but did not enter the inguinal canal. Apparently the arrangement of the abdominal muscles forming the walls of the inguinal canal prevents the ready passage of fluid along the spermatic cord or round ligament of the uterus through the inguinal canal into the scrotum or labia.

The other case is from an autopsy performed in the Department of Pathology at this school. H. P., a 61 year old, white male, No. 52,684 had a reducible, inguinal hernia on the right side and complained of abdominal pain. Paracentesis abdominis before death demonstrated bloody serous, aspirated fluid. It was found at autopsy that the left gastroepiploic artery had a ruptured aneurysm about 7 centimeters from its origin on the splenic artery. Extravasated blood, in the form of clots, was present in most of the retroperitoneal space: from the posterior portion of the diaphragm, over the renal areas, along the ureters into the pelvis; in the mesentery of the small intestine (Fig. 23), wall of the duodenum, in the transverse mesocolon, and between the anterior and posterior layers of the omentum. Sections of the spermatic cord containing part of the herniated peritoneal sac (Fig. 24), contained extravasated blood external to the herniated peritoneal sac. The greater peritoneal cavity contained about 3000 cubic centimeters of serous bloody fluid and the lesser peritoneal cavity was filled with about 1000 cubic centimeters of clotted blood. This second case demonstrates the many possible pathways of extravasation from an artery of medium size. Apparently the extravasation first occurred into the retroperitoneal tissue and after rupture of the peritoneal surface, into the lesser omental cavity. From here the blood possibly spread through the foramen of Winslow into the greater peritoneal sac.

Croce, Johnson, and Wiper (1945) have shown that following perforation of the infraperitoneal portion of the rectum, feces contaminate the potential space around the rectum—mainly in a posterior direction, between the inner stratum around the rectum and the outer stratum (pelvic fascia) over the sacrum (Figs. 15 and 17). Fecal contamination spreads posteriorly over the sacrum and then ascends

between these strata resulting in widespread retroperitoneal sepsis.

DISCUSSION

In a dissection of fascial strata in a body free of pathology, cleavage planes are necessarily made which do not exist normally. Histological sections of any region containing several fascial strata show that all the strata are interconnected by areolar or adipose tissue which varies in amount with the mobility between the various strata and the obesity of the specimen. A study involving dissection of fascial strata therefore is difficult and is subject to criticism, particularly where embalmed material is used, for fixation necessarily changes the texture of the tissues. Similar criticism also is made of the use of fixed material for both normal histological and pathological studies, yet these methods certainly serve a useful purpose and will continue in use until better means of studying this material are available.

The use of embalmed material for the basic dissections was necessitated by an insufficiency of unembalmed material and especially since surgery involving such extensive areas in one individual would be prohibitive. Our findings on the layers in fixed specimens have been verified, however, in living subjects at operation, on deceased bodies at autopsy, and by clinical findings involving the fascia described. This study was planned primarily to ascertain the dissectible fascial strata of surgical importance and the potential spaces between these strata which infection or extravasation would follow.

Embryologically the fascia under consideration formed one continuous layer. With increasing age the derivatives of this one layer became further differentiated and could be dissected as several strata. This stratification could be followed quite easily in the abdomen; however, in the pelvis, where through change in position of the viscera and formation of pads of areolar and fatty tissue or local thickenings to form ligaments, the composition appears more complex. Yet traced from its embryological origin and the continuity of the dissectible strata in the adult, the pelvic layers appear to have the same form and relation as those of the abdomen, and by analogy, the strata of both regions can be compared to the layers on the outside of the body. The following analogy is proposed therefore to help clarify these concepts of the continuity of the fascia lining the abdomen, pelvis, and spermatic cord (Table I). On both the inside and outside of the framework (musculoligamentous-bony) of the body the following comparable layers are found:

1. Surface epithelium. The mesothelium of the peritoneum forms a continuous layer lining the abdominal cavity just as the epidermis of the skin forms a continuous layer for the outside of the body.

2. Supporting tissue of epithelium. The connective tissue immediately subjacent to the mesothelium of the peritoneum forms a supporting layer for this mesothelium just as the dermis of the skin supports the epidermis.

3. Retroperitoneal and subcutaneous tissues. Subjacent to the connective tissue supporting the mesothelium is the retroperitoneal tissue, on the inside of the body, and the subcutaneous fascia beneath the dermis on the outside of the body. Both of these layers have many common features. Their texture varies in different parts of the body under consideration, being areolar, fatty, collagenous, or elastic in varying proportions; depending on the mobility of the surface layers and the degree of protection and support afforded to the area. These layers may be in the form of more than one lamina depending on the body type of the individual: the retroperitoneal tissue may form two laminae in the region of the kidneys and ureters, and adjacent to the inguinal region; just as Camper's and Scarpa's fascia form two laminae over the ventral abdominal wall. Many structures are embedded in, or course through, these analogous layers: the viscera and blood vessels are embedded in the retroperitoneal tissue, whereas the subcutaneous tissue contains part of the sweat and mammary glands, hair follicles, blood vessels, nerves, lymphatics, etc. The testes, epididymides, vasa deferentia, and their vessels and nerves, originally embedded in the retroperitoneal tissue while in the abdominopelvic cavity, are embedded in a continuation of the retroperitoneal tissue through the inguinal

canal into the scrotum of the adult. In both cases smooth muscle may be embedded in this layer: the muscle in Denonvilliers' fascia, puboprostatic ligament, etc. for the retroperitoneal layer, and the dartos, corrugator cutis, etc. for the outer layer.

4. Deep fascia. Beneath the subcutaneous and retroperitoneal tissue is a thickened collagenous and elastic layer which ensheaths and binds together the various components (muscles, ligaments, and bones) of the body wall. This layer is known as the transversalis fascia on the inside of the body wall and the deep (muscular) fascia on the outside of the body wall. As Hertzler (1913) has aptly stated: "The individual is interposed between a cutaneous and a serous sac."

Through conventional usage the terms visceral and parietal have become applied to the peritoneum and particularly to the endopelvic fascia. For descriptive purposes such terms may have a rôle, but anatomically there are areas where the dividing line between parietal and visceral layers, or vice versa, cannot be made, for these layers are continuous.

Diaphragm. The connective tissue on the caudal surface of the diaphragm is usually described as one layer. However, if the continuity of the fascia around the structures connected to, or perforating, the diaphragm are traced caudad, representations of all three strata can be found on the diaphragm. The outer stratum is attached to the periphery of the diaphragm. The laminae of the intermediate stratum cover its caudal surface where the adrenals and kidneys are adjacent to the diaphragm or the larger vessels pass through their diaphragmatic openings. The inner stratum blends with the other strata at the esophageal hiatus or the attachment of the liver to the diaphragm. All three strata blend to form one layer, except where there is movement between the diaphragm and the viscera; in such instances, the stratification remains.

Abdomen. The inner stratum around the gastrointestinal system does not have a parietal representation except for the connective tissue along the course of the vessels and nerves to this system, which connect the intermediate stratum to the inner stratum. The intermediate stratum should be considered visceral only in those areas where it embeds viscera: i.e., kidneys, adrenals, ureters, bladder, reproductive organs, vessels, and nerves. The outer stratum remains parietal in its entire extent.

Pelvis. The use of the terms parietal and visceral layers of the endopelvic fascia usually do not consider the continuation of these fascial layers cephalad into the abdomen. The plan of stratification of the abdominal and pelvic fascia described in the preceding part of this paper suggests a better working description of the pelvic fascia: an outer stratum for the bones and muscles of the pelvic wall and floor (iliac, obturator, levator ani, and sacral fascia, etc.); an intermediate stratum around the pelvic urogenital organs together with their vessels and nerves; and an inner stratum around the sigmoid colon and rectum. These fascia interconnect where the viscera perforate the pelvic diaphragm or along the course of the pelvic vessels and nerves.

The complexity of the derivatives of one continuous layer in the embryo, which in the adult forms several strata, can be well illustrated by the connective tissue around the bladder. Connective tissue adjacent to the peritoneum forms part of the covering for the superior surface of the bladder. In addition to this, there is a fairly dense layer associated with the bladder musculature termed the "collar of the bladder." This collar is continuous with other fascial strata: the connective tissue along the course of the ureter, that derived from Denonvilliers' fascia, the dense connective tissue derived from the levatori ani fascia (the collar of the bladder), the fatty and areolar tissue in the space of Retzius, together with the same type of tissue around the vas deferens which forms part of the spermatic cord.

The authors agree with Curtis, Anson, and Beaton (1940) that many of the so called ligaments, even though termed false ligaments are not supporting structures in any sense of the term—such as the peritoneal folds, termed median and lateral umbilical ligaments and sacrogenital ligaments. If these ligaments have any supportive function, it is the nature of the tissue subjacent to the peritoneum which is the supportive element.

Congdon (1937) suggests a functional explanation for the differences in the structure of the fascia in the various parts of the abdomen and pelvis. For example, structures like the fascial collars around the bladder and rectum could be due to thickenings in their connective tissue coverings resulting from the functional distention of the viscera; or the renal fascia could be considered as a localized thickening of the intermediate stratum due to the growth of an organ within the original mesenchyme. Wherever there is movement between viscera, an interposed shearing or frictional layer is found between the fasciae embedding the viscera. Examples of this type of fascia are found in the connective tissue of the space of Retzius (between the bladder collar and pelvic floor and wall) and the dorsal lamina of the intermediate stratum (between the ventral lamina embedding the adrenals, kidneys, and ureters and the outer stratum of the abdominal wall).

Many of the concepts suggested in this paper are not original. An attempt was made to correct some of the older concepts; correlate previous studies with the findings presented; reinterpret former work in view of more recent findings; and to suggest the possible clinical value of the fasciae in the abdomen, pelvis, and spermatic cord.

SUMMARY

1. The fasciae lining the abdomen, pelvis, and spermatic cord form continuous layers. These fasciae are dissectible into 3 main strata: (a) an inner stratum for the gastrointestinal system; (b) an intermediate stratum embedding the urogenital system, adrenals, larger vessels, and nerves; and (c) an outer stratum, the transversalis fascia, which is the thickened fascia of the musculoaponeurotic-bony framework of the body wall. The intermediate stratum may be in the form of two laminae in the region of the kidneys and ureters and adjacent to the ventral abdominal wall.

2. Cooper's original definition of the term transversalis fascia is reviewed and the erroneous definition of this term, given in many of the current texts, is discussed. The transversalis fascia is not limited to the inner surface of the transversus abdominis muscle but is continuous with the intrinsic fascia of the other components of the abdominal and pelvic wall.

3. The internal spermatic fascia appears not to be a distinct fascial layer similar to the external spermatic or cremasteric layers in individuals without indirect inguinal hernia. This restudy indicates that it is the areolar and fatty tissue, embedding the components of the spermatic cord, which is continuous with the retroperitoneal tissue of the abdomen and pelvis.

4. Clinically these strata are of importance: (a) as surgical guides in the abdomen, pelvis and spermatic cord; (b) as a barrier to, or pathway for, the spread of infection, of extravasated blood or urine, or injected gases for insufflation studies of the renal-adrenal region; (c) the retroperitoneal tissue embedding the ureters may be involved in the production of ureteric obstruction.

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TRANSURETHRAL RESECTION IN THE SURGICAL MANAGEMENT OF CORD BLADDER

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IT appears that we are at last slowly finding our way out of the hopeless confusion of the problem of the cord bladder. We may not be extricating ourselves in the purely scientific manner which we had desired but, after all, in the last analysis the end of such a medical quest is relief for the patient who suffers from the disease. We are now relieving the urinary troubles of many patients who have cord bladder, in spite of the fact that we still are unable to answer correctly and completely all questions concerning the neurophysiology and mechanism of micturition. For the present, at least, the advances in this field are being made by "cut and try" therapeutic procedures, which, in addition to helping the patient, are throwing a new light on the causative factors involved in the vesical dysfunction that results from inflammatory, degenerative or traumatic lesions of the central nervous system, and especially those of the spinal cord.

A voluminous literature attests to the fact that much experimental work has been done to determine the neurophysiology of the normal bladder, as well as that of the abnormal bladder resulting from neurologic trauma and disease. That this literature is contradictory and for the most part unenlightening is shown by comprehensive reviews of workers (12) who have attempted to correlate it. A critical analysis, however, shows that some of the experimental work has been done accurately and has been corroborated by several careful investigators. Based on the results of these studies, certain more or less accepted theories as to the neurophysiology of the normal and of the cord bladder have been postulated.

THE NEUROMECHANICS OF MICTURITION

The theory of the antagonistic innervation of the bladder by the sympathetic and parasympathetic nerves has lost most of its proponents. At present, it is supposed that the sympathetic supply to the bladder (the hypogastric or presacral nerve, which originates in the eleventh and twelfth thoracic and the first lumbar segments of the spinal cord) has little to do with control of the bladder. It may contain some sensory fibers which carry pain and sense of temperature. Also, the control of the blood vessels in the wall of the bladder exercised by the sympathetic fibers may be of some importance.

The important nerve supply of the bladder seems to be parasympathetic (nervi erigentes, or pelvic nerves, which originate in the second, third and fourth sacral segments of the spinal cord). These nerves carry both sensory and motor fibers, and apparently form the primary nerve supply for control of the urinary bladder.

The pudendal nerve (somatic, which originates in the second, third and fourth sacral segments of the spinal cord) no doubt plays a definite rôle in the control of micturition. Just how important a rôle this is, however, is open to question. This nerve supplies the external urethral sphincter (compressor urethrae muscle), which is supposed to be a semivoluntary muscle.

Munro very aptly expressed the currently accepted neurologic explanation of the mechanics of micturition when he stated that it is a "process of inhibition rather than one of facilitation." As Denny-Brown and Robertson (2, 3) have said, micturition is the reflex response of the muscle fibers of the detrusor urinae to stretching. The reflex center is thought to be located in the sacral portion of the spinal cord. As the bladder distends by being filled with urine, its natural response is to contract and to expel its contents. This action is prevented, however, by subconscious inhibitory impulses from the brain which act on the central synapses of the reflex arc in the lateral horns of the sacral part of the spinal

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cord. As the bladder becomes more distended, the sensory stimuli become more marked and reach the threshold of consciousness in the form of the desire to void. If the urge to void is great, it may be necessary for the brain to send down additional voluntary inhibitory impulses to the reflex center in the sacral part of the spinal cord. It may be necessary for the individual to send down voluntary impulses to cause a voluntary contraction of the external sphincter and perineal muscles. Such an action is stated to cause a "reflex" relaxation of the bladder. When the individual wishes to pass urine he then "inhibits" the inhibitory impulses and allows the detrusor urinae to contract and expel the urine. The existence of a second reflex mechanism has been postulated. It is asserted that when urine enters the prostatic part of the urethra, a reflex is initiated by way of the pudendal nerve and sacral part of the spinal cord which causes the external sphincter muscle to relax.

The pathways in the spinal cord which have to do with the cerebral control of micturition are not too well understood. It seems definite that there are no motor impulses which proceed from the cerebral cortex to the pelvic nerves (nervi erigentes) to initiate contraction of the bladder. The inhibitory impulses which arise in the brain at a subconscious level proceed to the central synapses (of the reflex arc for micturition) in the lateral horns of the sacral part of the spinal cord. The exact path of these impulses is not entirely clear. The motor impulses from the cerebral cortex pass by way of the pyramidal tracts to the pudendal nerve cells in the anterior horn of the spinal cord in the sacral portion to produce voluntary contraction of the external sphincter muscle. The sensory impulses arising in the bladder for the most part pass by way of the posterior roots of the pelvic nerves or nervi erigentes to the posterior horns of sacral part of spinal cord. According to Barrington (1932), these impulses then cross to the opposite lateral column (spinothalamic tract).

THE "URINARY SPHINCTERS"

There is much confusion concerning the sphincters which keep the urine confined to the bladder. For years workers have spoken of an "internal" and an "external" sphincter. Lewis emphasized the fact that there is no true internal sphincter. The vesical neck apparently is composed of the edge of the detrusor urinae muscle, which is able to contract to a considerable degree. There is no doubt that the normal prostatic urethra has the ability to contract to a certain degree. It is stated by some workers that the vesical neck is the greatest single factor in the control of urination. It is true that cystograms of most patients who have normal bladders show the medium confined to the bladder and apparently not entering the prostatic urethra. For further proof, the aforementioned workers cite the facts that after perineal prostatectomy, or when the external urethral sphincter has been damaged by trauma, control of urine may still be perfect by virtue of a competent vesical neck or "internal sphincter."

On the other hand, the experience of the present writers in several thousand cases of transurethral prostatic resection suggests that the all important sphincter is the external sphincter. In the cold-punch type of resection which is done at the Mayo Clinic no attempt whatever is made to preserve the normal vesical neck or "internal sphincter." Nevertheless, if the area of resection is kept proximal to the region of the external sphincter and if this sphincter is not damaged, urinary control remains perfect and micturition is normal in every way. If the external sphincter is cut, however, incontinence always ensues. This situation would seem to question the validity of the postulate that there is a second reflex in micturition; namely, the one which occurs by way of the pudendal nerves, and effects relaxation of the external sphincter and perineal muscles when urine first enters the prostatic urethra. We feel reasonably sure that in some cases, after transurethral resection, the vesical neck (including the edge of the detrusor urinae muscle) is resected so widely that it would be entirely unable to act as a sphincter mechanism and to prevent urine from entering the prostatic urethra. In some cases, cystograms made after transurethral resection tend to substantiate this assertion, because in these cystograms the medium is seen filling the bladder as well as the widely open prostatic

urethra. In spite of this, the bladder of the patient is continent and the patient is unaware of any abnormality in the act of micturition. Also, the experience of the present writers in observing the vesical neck of many patients from above by cystoscopy through incisions made at suprapubic cystostomy leaves them with the impression that the vesical neck is not tightly closed. At least, it does not seem to be sufficiently tight to keep the prostatic urethra entirely free of urine.

This latter reasoning would suggest that the external sphincter is the most important single factor in the control of micturition. Such an assertion raises some problems which are difficult to explain. As Lewis has aptly stated, "The voluntary sphincter is not adapted anatomically for retention of urine. Striated musculature has not the endurance for continuous maintenance of tone which is the great attribute of smooth muscle." In spite of the apparently sound reasoning of Lewis, practical experience suggests that the normal status of the external sphincter is one of tonic contraction. For instance, if an attempt is made to inject fluid in a retrograde manner through the urethra, some difficulty is encountered, and pressure is required to force it beyond external sphincter. X-ray studies made with opaque medium make it clear that the point at which the fluid is "held up" is the external sphincter and not the vesical neck.

The conclusions to be drawn from this brief discussion of the "urinary sphincters" are not too enlightening. At best, about all that can be said is that the last word on this subject has not been spoken. Future investigative work may throw more light on this poorly understood subject, and allow an accurate answer to a most perplexing problem.

RESULTS OF EXPERIMENTAL LESIONS OF THE SPINAL CORD

As mentioned previously herein, it is difficult to evaluate the literature concerning vesical dysfunction that results from experimental lesions of the spinal cord. The most nearly accurate work which has been more or less corroborated by careful investigators suggests that certain statements are true (13). Experimentally, the reflex arc for micturition may be abolished and the bladder separated from the control of the central nervous system by (1) section of both pelvic nerves (nervi erigentes), (2) section of the anterior roots of both pelvic nerves, (3) destruction of the cauda equina, or (4) destruction of the conus medullaris (which contains the sacral segments of the spinal cord.) Any one of these lesions results, after the shock phase of vesical atony has passed, in an "autonomous type" bladder which is characterized by (1) increased tone, (2) decreased capacity, (3) hypertrophy of the vesical wall, (4) active "incontinence," (5) the presence of frequent, weak, irregular, inefficient vesical contractions, (6) the presence of residual urine and (7) in some cases, evidence of dilatation of the upper part of the urinary tract. When this condition has been produced in the bladder, there are frequent uncontrolled spurts of small amounts of urine which keep the animal continually wet.

If the lesion is made above the sacral segments of the spinal cord, so that the reflex center for micturition remains intact, the result usually is an "automatic type" of bladder which is characterized by (1) increased tone, (2) decreased capacity, (3) active "incontinence," (4) the presence of reflex vesical contractions, and (5) the presence of residual urine. This type of bladder differs from the "autonomous" in that it tends to be more efficient, the interval between evacuations of urine is longer, and the amount of residual urine usually is less. The bladder is not so tonic, and the detrusor urinae muscle does not become so thickened, as in the "autonomous" bladder.

CLINICAL ASPECTS OF INJURY TO THE SPINAL CORD

Classification. Difficulty is encountered when an attempt is made to apply the observations made in the experimental laboratory to clinical lesions encountered among patients. In the first place, in clinical practice the urologist seldom sees the definite, complete, clean-cut lesions which are produced experimentally. Although in most cases it is easy to determine the upper level of the lesion, it may be most difficult to determine the exact condition of the spinal cord below this level.

Damage to the cord may have a patchy distribution which makes evaluation of the true condition extremely difficult. It is also important to realize the relationship of the several segments of the cord to the vertebrae, because all the lumbar and sacral segments of the spinal cord are confined to an area bounded by the eleventh and twelfth thoracic vertebrae and first lumbar vertebra. The possibilities of damage to spinal cord and cauda equina by trauma in this area can be seen.

There have been many attempts to classify cord bladder. The actual value of such classification is questionable. One of the best and most complete classifications is that of Munro. He recognized five chief divisions; namely, (1) atonic (shock bladder), (2) autonomous, (3) hypertonic, (4) uninhibited reflex, and (5) so called normal cord bladder. Munro is in agreement with most investigators of this subject when he states that the "autonomous" is the best vesical condition that can be expected to remain if the reflex arc for micturition has been completely destroyed by a lesion of the sacral portion of the spinal cord or cauda equina. Lesions sustained above this level may permit establishment of one of the more efficient types of cord bladder enumerated in Munro's classification, depending on level of the lesion and degree of recovery.

In the experience of the present writers, the clinical classification of cord bladder is very difficult and is of questionable value. In such a consideration the atonic bladder should be excluded. It is encountered in the presence of two conditions: (1) immediately after injury to the spinal cord, regardless of the level of the lesion, and in this case it is known as the "shock stage," and (2) as a result of lesions of the posterior roots of the pelvic nerves, which lesions are seen almost exclusively in the presence of tabes dorsalis.

In this paper we are not concerned with the acute phase of injury to the spinal cord, but rather, with the chronic stage which is present weeks or months after the injury. Also, we shall not consider the so-called tabetic bladder, because its treatment has been fully discussed elsewhere (5, 11), and is no longer a urologic problem of any importance. The problem at hand, therefore, is the chronic stage of "true" cord bladder which would have a place somewhere in Munro's classification of autonomous, hypertonic, uninhibited reflex and so-called normal cord bladder.

On the basis of our experience, the most practical attitude to assume is one in which two extremes of cord bladder are recognized. On the one hand is the most inefficient type of cord bladder, called the "autonomous." At the other extreme is the most efficient type of cord bladder, which may be designated as the "automatic" type. This roughly corresponds to Munro's "hypertonic" or "uninhibited reflex" type of bladder. The autonomous type of bladder represents the condition which obtains when severe damage to the sacral part of the spinal cord and the reflex arc has occurred. It is a bladder which will evacuate frequent, small spurts of urine, keeping the patient continually wet, but which will always contain a large amount of infected residual urine. The automatic type of bladder, on the other hand, is one in which fairly long intervals (up to 3 or 4 hours) may prevail between evacuations of urine. The amount of urine evacuated at each voiding may be considerable, and the residual urine usually is of smaller amount than that of the autonomous type of bladder. Also, the patient may have some warning that evacuation is about to occur, so that sufficient time is allowed in which to procure a urinal to prevent the patient from soiling himself.

Conventional therapeutic aspects. Therapeutic aspects of the chronic stage of cord bladder in the past have been rather negative. Results of therapy have been even more discouraging. So far as the autonomous type of bladder is concerned, about all that has been done is to teach the patient to employ abdominal straining (if the abdominal muscles are intact) and manual compression of the bladder. This, if done at intervals, would tend to keep the patient dry for longer periods. An indwelling urethral catheter or the constant use of a urinal was the only alternative. Neither of these measures allowed eradication of the urinary infection. The automatic type of bladder was managed a little more satisfactorily. A patient with such a condition could be taught to find a "trigger area"

somewhere on the body, stimulation of which might initiate micturition. In the more efficient automatic type of bladder, this procedure proved to be fairly satisfactory. Knowing the approximate length of time elapsing between voidings, patient could anticipate the occurrence of micturition, and could initiate it by stimulating trigger area. The constant presence of residual urine, however, defeated most attempts to eradicate the urinary infection.

Newer therapeutic aspects. During the last decade there has been a gradual evolution in thought concerning the problem of cord bladder (16). To follow the line of reasoning involved, it is necessary to understand a few basic concepts of this condition. There are certain common characteristics of all cord bladders (if lesions of only the sensory portion of the reflex arc are excluded). These are (1) increased tone of the bladder, (2) presence of residual urine, and (3) "active" incontinence of the same type.

In every case of true cord bladder there are *increased tonicity and increased thickness of the detrusor urinae muscle.* In some cases this condition is extremely marked, resulting in a "spastic" bladder. Observations suggest that the "autonomous" or least efficient type of bladder may be the seat of greater tonicity and hypertrophy of the detrusor urinae muscle than the more efficient "automatic" type of bladder. *Trabeculation* is a prominent finding in all cases of cord bladder. The degree of trabeculation or caliber of the trabeculae varies greatly.

As stated previously, in all (or nearly all) cases of true cord bladder there is *residual urine.* Munro has expressed the values for residual urine in terms of per cent of the fill (vesical capacity). Expressed in this manner, the "autonomous" type of bladder carries a greater percentage of residual urine than does the more efficient "automatic" type of bladder. No matter how efficient a cord bladder becomes, however, it almost never is able to eliminate its contents completely.

"*Incontinence*" is a term badly used in the urologic literature. It has been used to describe every type of condition, from complete passive urinary leakage to marked "urgency" of urination. In most cases of true cord bladder incontinence is not manifested by a passive, continuous dribble of urine similar to that which is noted when the external urethral sphincter has been destroyed. Rather, it consists of intermittent spurts of urine which are evacuated by more or less inefficient vesical contractions. This evacuation of urine varies from frequent spurts of small amounts by the inefficient "autonomous" type bladder to the sudden precipitate evacuation of more substantial amounts of urine with little or no warning at reasonably long intervals by the more efficient reflex vesical contraction of the "automatic" type of bladder.

Inasmuch as a cord bladder has increased tone and muscular strength, it never has been entirely clear why such a bladder should not be able to perform a better job of evacuating its contents than it does. The best explanation available supposes the reason to be the inefficiency of the vesical contractions. This is especially true of the "autonomous" type bladder which is completely severed from nervous connection with the spinal cord, so that reflex contractions no longer occur, but rather, "autonomous" rhythmical contractions are initiated, supposedly by reflex centers within the wall of the bladder itself. The persistence of residual urine is still more difficult to explain in the case of the "automatic" type of bladder, in which the reflex arcs are intact. To assist in the explanation, the analogy of the infant's bladder has been given. For instance, in the case of the newborn infant there is no voluntary control over micturition. Micturition is entirely reflex in type, yet the bladder empties its contents completely at each evacuation. To account for this, it is postulated that there is a center in the brain stem, the presence of which is necessary to cause the bladder to contract sufficiently to expel its contents completely. In the infant, whose pyramidal tracts are unmyelinated, the so called reflex type of bladder empties completely because the aforementioned center in the brain stem is intact, and the impulses can get through extrapyramidally. In the patient who has had spinal cord transection, it is supposed that these impulses cannot get through.

A more attractive and plausible theory suggests that the vesical neck (which, as said pre-

viously, is the edge of the detrusor urinae muscle), also taking part in the general spasticity and hypertrophy of the vesical wall, itself becomes obstructive. This would account, in part at least, for the residual urine. If this were true, removal of this obstruction should tend to eliminate the residual urine.

Clinical experience. The joint urologic experience of the present writers in this field has extended over 8 to 10 years, and roughly parallels their experience with transurethral resection. This experience can be described best in more or less narrative form with a few reports of illustrative cases. Our first observation of importance concerned patients who had sustained injuries to the spinal cord and who had achieved sufficient recovery to become ambulatory with the use of crutches or canes or, in some cases, with no help. In nearly all these patients a typical spastic type of gait persisted. In most cases the chief reason for complaint was vesical dysfunction, which was loosely described as "incontinence." When properly evaluated, this "incontinence" usually proved to be a precipitate type of active urinary incontinence which would occur at varying intervals, with little or no warning. The condition was sufficiently disabling to require patient to wear a urinal to avoid soiling. Another common reason for complaint was recurring episodes of acute urinary infection.

In most of these cases examination disclosed infection of the urine. In some cases an excretory urogram revealed hydronephrosis. In all cases the passage of a catheter after micturition had occurred demonstrated the presence of substantial quantities of residual urine. It was also noticed that after catheterization the patient experienced relief of his "incontinence" for several hours, until a quantity of urine equal to his "residual urine" had accumulated in the bladder. It was also seen on cystoscopic examination that this type of bladder was markedly trabeculated, had increased tone and varying degrees of fixation, and contraction of the vesical neck which appeared to be obstructive. We reasoned that removal of this obstruction might eliminate the residual urine, and thereby restore vesical function to a more nearly normal state. Hence, transurethral resection of the vesical neck was cautiously performed (6). A case in point is illustrative.

First illustrative case. A white man, 36 years old, was admitted to the Mayo Clinic on February 18, 1937. Nine years previous to his admittance he had sustained fracture of a vertebra in an automobile accident. Complete paralysis of the lower extremities and complete retention of urine had resulted. The patient gradually had recovered some degree of control of his lower extremities, but had continued to employ routine catheterization for 7 years.

At the time of admission to the clinic, the patient was able to walk with a cane. His chief reason for complaint was marked urgency and frequency of urination, with partial urinary incontinence. Incontinence consisted of precipitate urination before the patient was fully aware of it, and could reach the toilet. Neurologic examination revealed partial paralysis of lower extremities, with marked diminution in sensation of touch and pain. Lesion was at level of twelfth thoracic segment of spinal cord.

Cystoscopy disclosed trabeculation of the bladder, grade 1 on a grading basis of 1 to 4, in which 1 represents the least and 4 the most trabeculation. About 300 cubic centimeters of residual urine was present. It was noticed that after the bladder had been emptied with a catheter, the patient would remain dry and would not experience frequency or precipitate urination for at least 5 hours. Transurethral prostatic resection was performed on February 23, 1937. Three grams of tissue were removed. The pathologist reported this material to be smooth muscle and inflamed tissue. The result of this procedure was excellent. The patient was able to void a satisfactory stream of urine and had neither residual urine nor more urinary incontinence.

About this same time some encouraging results were being obtained by means of transurethral resection among infants and children suffering from urinary retention and incontinence. Many of these children had associated spina bifida, with or without other neurologic manifestations. This interesting phase of the subject was reported on by Thompson and Jacobson (19).

After experience with several more adult patients who had injuries to the spinal cord, we became convinced that many such patients could be relieved of their distressing urinary symptoms by means of transurethral resection. Up to this time, however, we had confined our work to patients who had achieved sufficient recovery from the injuries to their cords to be ambulatory. Whether or not transurethral resection would be of value for nonambulatory patients was not known. In

the summer of 1942 one of us (Emmett) performed transurethral resection for a bedridden patient who had sustained a traumatic injury to the spinal cord. Results were encouraging, as is detailed in the report which follows.

Second illustrative case. A physician, 36 years old, had been injured in an automobile accident on November 10, 1941, and had suffered fracture in the thoracic portion of the spinal column, with complete paralysis of the lower extremities. He had gradually regained a slight degree of voluntary movement in the right leg. The patient had become bedridden. A urethral catheter had been inserted, but later on, suprapubic cystostomy had been carried out because of acute urinary infection and vesical calculi. The suprapubic incision had been allowed to close after 3 months.

After this, extensive urinary infection, with purulent urine and the passage of calculi, had occurred. Repeated attacks of chills and fever had supervened. Three weeks before admittance of the patient to the Mayo Clinic, secondary suprapubic cystostomy had been done elsewhere. The tube had come out shortly before admittance of the patient to the clinic, and most of the urine had been voided by way of the urethra. Neurologic examination disclosed spastic paraplegia of the lower extremities, except for a slight degree of voluntary motion in the right leg. The sensory level did not extend below the ninth thoracic segment, although some irregularly distributed areas in which tactile sense remained were noted in some places below this level. An excretory urogram revealed the kidneys to be urographically normal. The patient expelled small amounts of urine every 30 to 45 minutes. There was 300 cubic centimeters of purulent residual urine. The patient experienced some slight warning of the imminence of micturition, but he had to keep a urinal in place constantly to keep dry. Cystoscopy disclosed trabeculation of grade 2 plus, and moderate increased tonicity of the bladder and vesical neck. Several soft, flat stones lay free in the bladder; these were readily broken up with the blunt specimen forceps and removed.

The primary problem in the condition of this patient was to secure elimination of residual urine so that the urinary infection could be eradicated. The secondary problem was to improve urinary control and to lengthen the interval between evacuations of urine. The patient gave his consent to performance of transurethral resection. This operation was carried out on July 25, 1942. Five grams of tissue were removed. The pathologists' report was "adenofibromatous hyperplasia." The result of this procedure was that, by means of abdominal straining, the patient was able to empty the bladder, except for a residual amount of urine which varied between 30 and 45 cubic centimeters. The patient could retain the urine for 2 hour intervals and keep dry. This greatly eased the nursing problem. The patient was treated in the Section on Physical Medicine for about 2 months, and at the time of his dismissal he had pyuria of only grade 1.

It is apparent from reading the foregoing illustrative case that the results of transurethral resection seemed to justify the effort. The nursing problem was greatly simplified and the patient could be kept dry.

At the meeting of the American Urological Association in St. Louis in June, 1944, one of us (Emmett) reported his experience at the Mayo Clinic with transurethral resection for cord bladder up to January 1, 1944 (9). Twenty cases of true cord bladder in which transurethral resection had been carried out were reported. In 13 transverse lesions of the spinal cord had been caused by trauma or inflammation. Results were excellent in 8 cases. In 2 cases the condition of the patients was improved, and in 3 cases it was not improved

TRANSURETHRAL RESECTION IN 45 CASES OF CORD BLADDER FROM JANUARY 1, 1944, TO JANUARY 1, 1946[1]

At the Mayo Clinic during the 2 year period, January 1, 1944, to January 1, 1946, transurethral resection was carried out for 45 patients who had neurogenic bladder resulting from injury or disease of the spinal cord. Thirty of these patients were suffering from a transverse lesion of the spinal cord, whereas the neurologic disease of the remaining 15 consisted of multiple sclerosis (7 patients), combined sclerosis (4 patients), and miscellaneous lesions of the spinal cord (4 patients). The first group (30 patients) will be considered now and the second group (15 patients), later. Twelve illustrative cases will be presented as the paper proceeds, in addition to the 2 presented previously herein.

Data concerning 30 patients who had transverse lesions of the spinal cord. Causation.—The cases of most interest are those involving the 30 patients who had transverse lesions of the spinal cord. From an etiologic standpoint, the lesions of 17 patients were the results of trauma (fracture of the spinal column, gunshot wounds, and so on), 6 lesions followed inflammatory myelitis, 3 were the sequelae of tumors of spinal cord which had been operated

[1]One patient treated in 1945 was still under treatment at the time this paper was written.

on and 4 lesions occurred secondarily to previous administration of spinal anesthesia.

Sex.—Twenty-five patients were males and 5 were females.

Age.—The ages of the patients varied from 14 to 69 years. Twenty-two of the 30 patients were 45 years old or less.

Duration of the lesion.—The interval of time which elapsed since the lesion to the spinal cord had occurred varied from 4 months to 27 years. In 18 cases it was 5 years or less. In 13 cases it was 2 years or less.

Level of the lesion in the spinal cord.—The level of the lesion in the spinal cord was as follows: in the cervical portion, 3 cases; in the upper thoracic portion, 2 cases; in the lower thoracic portion, 6 cases; in lumbar portion, 5 cases; in conus medullaris or cauda equina, 12 cases; widespread injury to cord, 1 case; level of lesion indefinite, 1 case.

Degree of recovery from paralysis.—Ten of the 30 patients were bedridden. Three were ambulatory with the use of crutches or canes, and 17 had obtained recovery sufficient for them to be able to walk without the help of canes or crutches. The type of gait employed by these 17 patients varied in degree from a pronounced, difficult spastic gait to an almost normal gait among a few patients who had secured marked recovery.

Residual urine.—The amount of residual urine varied from 50 to 1,200 cubic centimeters. The residual urine of all but 2 patients was 100 cubic centimeters or more.

Condition of the upper urinary tract (as determined by excretory urography).—Unfortunately, an excretory urogram was not obtained in all these cases prior to transurethral resection. An excretory urogram was made for only 17 patients. Four of these 17 patients were shown to have varying degrees of bilateral pyeloureterectasis, 1 was seen to have unilateral ureterectasis, whereas the remaining 12 were entirely normal, so far as excretory urograms were concerned.

Type of vesical dysfunction and correlation with site of lesion in spinal cord.—Generally speaking, it was practically impossible in this group of 30 patients to make any correlation between the type of vesical dysfunction and the site of the lesion in the spinal cord. It may be that the reason for this is that the group is too small to have any substantial number of lesions appear at any one level. As the literature accumulates on this subject, and especially when the information from the medical departments of the armed forces becomes available, definite correlation may be possible. The vesical symptoms exhibited by our group of patients varied tremendously. In some cases there was complete retention of urine with overflow dribbling, the patient experiencing no desire or discomfort until the bladder was markedly distended and palpable above the umbilicus. At this stage varying degrees of discomfort would be noted, accompanied by overflow dribbling of urine.

The type of vesical dysfunction most frequently seen among the patients who had complete or almost complete paralysis seemed to be that in which more or less frequent emissions of varying amounts of urine took place without the patients' knowledge of the act. In some cases this may occur every 10 to 20 minutes, whereas in others the interval may be an hour or longer. In some of the cases in which there was a 1 to 3 hour interval between evacuation of urine, the patient might pass, in addition, an occasional small spurt of only a few cubic centimeters of urine. This usually occurred near the time at which evacuation of the bladder was about to occur. In almost all these patients, if the bladder were emptied with a catheter, "voiding" or "leakage" of urine would not occur until an amount of urine equal to the patient's residual urine again had accumulated in the bladder. In most of these cases the residual urine varied from 200 to 500 cubic centimeters.

A rather characteristic type of vesical dysfunction is seen among patients who have achieved rather marked recovery from paralysis, so that they are ambulatory. This kind of dysfunction can be described as a precipitate type of urgency incontinence. The patient is suddenly seized with a desire to urinate. The basis of this desire may vary from a normal sensation to some odd "aura" such as an "ache" in the abdomen, or some type of paresthesia in the perineum or thighs. The patient is then unable to reach a lavatory before the urine is evacuated. Such patients

always have a substantial amount of residual urine; they describe their condition as urinary "incontinence" for which they are forced to wear a rubber urinal. The condition is doubly disturbing to the patient because there is not a sufficiently regular interval between micturition to allow him to be near a lavatory and anticipate the act.

The vesical dysfunction of patients whose cord injury is a result of administration of a spinal anesthetic agent is interesting. In some cases dysfunction amounts to complete retention of urine with inability to void, accompanied by pain when the bladder becomes distended. In other cases the patient is able to void, but considerable abdominal straining is required, and still the bladder cannot be emptied. In still other cases the disability is more like the precipitate type of urgency incontinence described previously herein.

Cystoscopic observations.—Trabeculation. The present writers never have seen an instance of true cord bladder (in which a sufficient length of time had elapsed for the patient to recover from the shock stage) not accompanied by trabeculation. The trabeculation varies greatly in degree, as do the trabeculae in caliber. The old idea that fine trabeculation denotes neurogenic bladder, and that coarse trabeculation signifies obstruction at the vesical neck, is entirely erroneous.

Tonicity of the bladder.—In the experience of the present writers, practically all true cord bladders exhibit an increase of tone in the detrusor urinae muscle. This, of course, is not true when only the sensory components of the reflex arc have been damaged (as is the case in tabes dorsalis) or in the occasional *rare* case in which, for some reason or other, the sensory components are primarily affected. One characteristic type of cystoscopic appearance of the true cord bladder is the more or less constant "writhing" appearance of the bladder, which appears constantly to be undergoing irregular contractile efforts which may make satisfactory distention with water through the cystoscope rather difficult.

Condition of the vesical neck.—If the vesical neck is kept under observation during these contractile efforts of the bladder, it will be noticed that the vesical neck also goes through more or less the same type of contraction and dilatation. When the condition has been of relatively long standing, the vesical neck may appear to be thick and hypertrophic, much the same as the rest of the bladder. If considerable infection has occurred, fibrosis and fixation also may be associated. In some cases the vesical neck appears to have a "collar" of tissue surrounding it, so that it has much the same appearance as a collar of adenomatous tissue. Among older patients, in addition, varying degrees of prostatic hyperplasia may be present. Massive hypertrophy of the interureteric bar also is a common observation.

In some cases, spasticity and hypertrophy of the bladder may be of only minimal degree, and it may be difficult to demonstrate a contracture or collar at the vesical neck. Thompson (18) has suggested that in these cases, if the bladder be well distended with water, a prominent contraction ring at the vesical neck will become apparent. If a contraction, spasticity or collar obstruction at the vesical neck is not demonstrable to the satisfaction of the cystoscopist, it is unimportant so far as diagnosis and treatment are concerned. Resection of the vesical neck will yield good results in spite of the cystoscopic appearance of the structures, and often the obstruction will become apparent only after resection has been started (7).

Transurethral resection of the vesical neck.—The surgeon who is experienced in transurethral prostatic surgery will have no particular difficulty in learning to resect the vesical neck of patients who have cord bladder. Technically, however, the procedure is a little different, since in most cases there is very little prostatic hyperplasia, so that new landmarks and danger signals must be learned. In the first place, it probably will be necessary to perform the operation in multiple stages in a fair percentage of cases. For instance, in the 30 cases under analysis, resection was done in one sitting in 21 cases, in two sittings in 5 cases, in three sittings in 2 cases and in four sittings in 2 cases. The total amount of tissue removed varied from 1 to 20 grams, and it is interesting to note that the patient from whom 20 grams of tissue was removed in four sittings was a man 25 years of age who had

sustained traumatic transection of the spinal cord at the level of the fifth cervical vertebra 9 years before the operation.

The technical points of resection may be of interest. All of the patients under consideration underwent resection with the Thompson cold punch. The object of the procedure is to remove as completely as possible the hypertrophic or spastic vesical neck (formerly called "internal sphincter") and any hypertrophied or normal prostatic tissue that also may be present. The difficulty in such a procedure is to be able to determine when resection has been carried deeply enough at the vesical neck to remove its contractile ability, and yet to avoid perforation of the bladder at this point. With increased experience, the surgeon will learn the depth to which he may cut, but in our opinion a number of operations will require a second or even third sitting to secure satisfactory results. There are several reasons for this statement. If the surgeon tries to achieve sufficiently complete removal of tissue at the first operation in every case, he will occasionally encounter bleeding from the edge of the bladder and from the prostatic capsule. Such bleeding is difficult to control, and even when controlled will tend to recur at intervals, for several days or weeks. Also, the chance of perforation of the vesical neck will be considerable.

For reasons not too easily explained, it is often possible to resect a vesical neck much more widely at a second operation than at the first. A "shift" of the tissues in the area of operation seems to occur, so that at a second sitting as much tissue can be removed as at the first operation, and it may be possible to remove more. Owing to disturbance of the blood supply, the tissue is more or less avascular, and lines of demarcation are more easily recognized than at the first sitting. At a second resection the operation may be carried safely to a depth which would have involved great risk at the first operation.

Among some patients we have cared for, it has been our impression that, in addition to hypertrophy of the vesical neck, hypertrophy of the vesical wall adjacent to the vesical neck also acts as an obstruction. The interureteric bar at times enlarges to a tremendous degree, and is definitely obstructive. Cautious removal of this bar and some of the hypertrophic vesical wall near the vesical neck (often in the anterior quadrant) may be necessary. Ordinarily, when the surgeon attempts to cut the muscle fibers of the normal bladder with the cold-punch knife during prostatic resection, the knife "jams" and will not cut cleanly. This constitutes a warning that enables the surgeon to avoid perforation of the bladder. In the case of the hypertrophic cord bladder, the vesical wall takes on a different character, and may be cut crisply and cleanly like prostatic tissue.

It must be emphasized that, in addition to the vesical neck, if any prostatic obstruction is present, the entire prostatic urethra must be well resected out to the level of the verumontanum and external sphincter. One of us (Emmett) has discussed completely the technic of the cold-punch type of resection elsewhere (8). Urinary incontinence will not ensue if the external sphincter is undamaged during the resection.

Results of transurethral resection.—The results of transurethral resection in the 30 cases under consideration are more than encouraging. A classification of the results is as follows: good, 23 cases; improved, 4 cases; fair, 2 cases; poor, 1 case. In the patient who obtained only a poor result, the external urethral sphincter might have been damaged during the operation, so that incontinence ensued. The bladder emptied completely, however. This patient also had suffered unusual complications from the injury to his spinal cord, among which was intractable pain in the back and legs which could not be controlled, even by the intraspinal injection of alcohol.

For 2 patients only fair results were obtained. One was a man 52 years old who had suffered from inflammatory myelitis for a year previous to admission to the clinic. He had recovered from the paralysis remarkably well, and was ambulatory (the lesion had involved the lumbar part of the spinal cord). His bladder contained 150 cubic centimeters of residual urine and he had great difficulty in voiding. The removal of 3 grams of tissue by transurethral resection allowed the patient to empty the bladder completely, but he still complained of an inadequate stream

and of some degree of frequency. Additional resection no doubt is indicated.

The second patient was a man 51 years old in whom urinary difficulty and residual urine of about 200 cubic centimeters had developed after a spinal anesthetic agent had been administered 13 years previously. Removal of 7 grams of tissue (the pathologic report was "hypertrophied muscle") reduced the amount of residual urine to 60 cubic centimeters, but voiding was still imperfect. A second resection no doubt is indicated in this case.

The general problem can be portrayed best by several illustrative cases.

Third illustrative case. This case represents long-standing injury of the cervical part of the spinal cord with almost complete recovery except for the bladder. A man 48 years old was admitted to the clinic on April 16, 1945. He had sustained fracture of the fifth cervical vertebra in 1922. Complete quadriplegia had ensued and had persisted for 30 days. Gradual recovery had followed, until the only residual effect was "numbness" and some degree of weakness in the legs and slight foot drop on the right. The patient was unable to ejaculate. We found the abdominal muscles to be normal; the gait was almost normal. The patient had difficulty in voiding; the stream was poor. Nocturia was present, and residual urine amounted to 250 cubic centimeters. Cystoscopy disclosed trabeculation of grade 1 plus, with typical collar or contracture of the vesical neck. Transurethral resection was performed on May 22, 1945, during which 3 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." The results of operation were good. The urinary stream was excellent. There was no residual urine. Vesical control was good.

Fourth illustrative case. The case involves a long-standing traumatic lesion of the conus medullaris and the cauda equina, with partial recovery of the patient. A man 44 years old was admitted to the clinic on September 26, 1945. He had sustained fracture of the first lumbar vertebra in 1934. Paralysis of the lower extremities had ensued. Gradual partial recovery had followed. At the time we saw him he walked with a markedly spastic gait, using canes. He had some secondary contractures and deformities of the lower extremities. Neurologic examination disclosed saddle anesthesia and varying degrees of anesthesia of patchy distribution below the knees. The sexual function was impaired. The abdominal muscles were normal. Urgency incontinence was marked. The patient voided about every 2 hours; the stream was poor and interrupted. The patient found it necessary to carry a bottle with him. Residual urine amounted to 120 cubic centimeters. Cystoscopy disclosed trabeculation of grade 3. Marked contracture and scarring of the vesical neck, with some slight degree of adenofibromatous hyperplasia, were noted. An excretory urogram disclosed nothing significant. Transurethral resection was performed on October 5, 1945, during which 9 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." The result of the operation was good. There was no residual urine; the stream was satisfactory. Control was good, although mild urgency of micturition persisted immediately after the operation.

Fifth illustrative case. The case involves a traumatic lesion of the conus medullaris in a woman, with good recovery except for the bladder. The patient, a woman 34 years old, was admitted to the clinic on October 9, 1945. She had sustained fracture of the first lumbar vertebra in 1939. Loss of control of the bladder and rectum and 50 per cent loss of sensation and motor power of the lower extremities had ensued. The motor and sensory disturbances had gradually disappeared, but disturbance of control of the bladder and rectum had continued. The patient complained of constant leakage of urine at night. She said she had an almost constant "bearing-down sensation" and a desire to urinate in the daytime. She passed only small amounts of urine. She catheterized herself often, and would obtain thereby more than 300 cubic centimeters of urine. After catheterization, she would remain comfortable and dry for 4 to 5 hours. Neurologic examination disclosed saddle anesthesia. The motor power in the legs was normal when we saw the patient. The abdominal muscles also were normal. Cystoscopy showed trabeculation of grade 3, 300 cubic centimeters of residual urine, increased expulsive force, and a very short urethra. The first stage of transurethral resection was performed on October 15, 1945, during which 17 pieces of tissue, weighing 2 grams, were removed. The operative result was poor. The second stage of transurethral resection was performed on October 24, 1945, during which 22 pieces of tissue, totaling 2 grams were removed. The pathologic report on tissue removed at both operations was "inflamed muscle." The operative result was good. The urinary stream was satisfactory; there was no residual urine. Nocturia was noticed once. Control was good.

Sixth illustrative case. This case represents injury of the lower thoracic portion of the spinal cord, with flaccid paralysis of the lower extremities. The patient had become bedridden, and had overflow incontinence. This man, 29 years old, was admitted to the clinic on August 1, 1944. Three years previous to his admission he had sustained a fall which caused fracture of the spinal column, with complete motor paralysis and loss of sensation in the lower extremities. The patient had had no control of bladder or bowels since the injury. Neurologic examination disclosed flaccid paralysis of the lower extremities. The sensory level was at the twelfth thoracic vertebra. The upper abdominal muscles were normal; the lower abdominal muscles had loss of strength of grade 2 minus. Urinary retention was complete.

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Sixth illustrative case. This case represents injury of the lower thoracic portion of the spinal cord, with flaccid paralysis of the lower extremities. The patient had become bedridden, and had overflow incontinence. This man, 29 years old, was admitted to the clinic on August 1, 1944. Three years previous to his admission he had sustained a fall which caused fracture of the spinal column, with complete motor paralysis and loss of sensation in the lower extremities. The patient had had no control of bladder or bowels since the injury. Neurologic examination disclosed flaccid paralysis of the lower extremities. The sensory level was at the twelfth thoracic vertebra. The upper abdominal muscles were normal; the lower abdominal muscles had loss of strength of grade 2 minus. Urinary retention was complete.

The bladder filled and distended until it could be palpated at the umbilicus; then overflow occurred, keeping the patient wet continually. Cystoscopic examination disclosed mild trabeculation and a wide vesical neck, but the vesical neck was definitely hypertonic. At first stage transurethral resection on August 21, 1944, 3 grams of tissue was removed. The pathological report was "adenofibromatous hyperplasia." There was no improvement after this operation. At second stage transurethral resection carried out on August 28, 1944, 4 grams of tissue was removed. The pathologic report was "adenofibromatous fibroma." The operative result was good. Improvement was slow but gradual. In answer to a follow-up letter sent to the patient 3 months after operation, he said he emptied the bladder completely, except for a residual amount of 10 to 20 cubic centimeters, voided every 2 hours, strained the abdominal muscles to void and had a satisfactory stream. He was able to keep perfectly dry.

Seventh illustrative case. This case represents inflammatory transverse myelitis, with a lesion in the lower thoracic portion of the spinal cord and spastic paralysis. The patient had become bedridden. A male 18 years old was admitted to the clinic on September 29, 1945. Inflammatory myelitis had occurred in November, 1943, resulting in complete paralysis of the lower extremities, complete retention of urine for 10 weeks, followed by "incontinence." Exploratory laminectomy had been performed shortly after the onset of the condition. Neurologic examination disclosed spastic paraplegia, with the sensory level at the tenth thoracic vertebra. The abdominal muscles were intact. Spurts of urine occurred every 15 to 30 minutes. The patient said he experienced an unusual sensation in the lower part of the abdomen when the bladder was full. He could express some amount of urine by manual compression, but a residual amount of 300 to 500 cubic centimeters remained. An excretory urogram disclosed nothing significant. Cystoscopy showed trabeculation of grade 2 plus. The detrusor urinae muscle was spastic and hypertonic. At first stage transurethral resection carried out on November 7, 1945, 5 grams of tissue was removed. The pathologic report was "adenofibromatous hyperplasia." At second stage transurethral resection performed on November 16, 1945, 6 grams of tissue was removed. The pathologic report was "inflamed muscle." The operative result was marked improvement. The patient voided every 1½ to 2 hours. He could initiate urination by slight digital dilatation of the anal sphincter. He was using abdominal straining, and emptied the bladder completely. He still lost 10 to 20 cubic centimeters of urine in few small spurts during a 20 minute period just before urination began.

Eighth illustrative case. This case represents a traumatic lesion of the lower thoracic part of the spinal cord with complete spastic paralysis. There was no recovery from paralysis of extremities. A man 27 years old was admitted to the clinic on June 22, 1945. He had been injured in a mine cave-in in May, 1944. Complete paralysis of the lower extremities had ensued. Laminectomy had been performed 24 hours later. Use of an indwelling urethral catheter for 2 months had been followed by severe urinary infection and bilateral epididymitis. Suprapubic cystostomy had been done. The patient arrived at the clinic with the suprapubic tube in place. Neurologic examination revealed spastic paraplegia. The sensory level was at the twelfth thoracic vertebra. A decubitus ulcer was noted over the sacrum. The abdominal muscles were normal. Urologic treatment consisted of removal of the suprapubic tube and insertion of a urethral catheter. The suprapubic wound closed in 5 days. The urethral catheter was then removed, and vesical function was observed. After catheterization, the patient could remain dry for about 1 hour without leakage; then frequent spurts of urine at irregular intervals would keep the patient wet. Residual urine amounted to 350 cubic centimeters. Cystoscopy disclosed trabeculation of grade 3. The detrusor urinae muscle was spastic, and the vesical neck was very spastic, with an apparent collar of tissue resulting from hyperplasia of muscle. At transurethral resection on August 14, 1945, 6 grams of tissue was removed. The pathologic report was "adenofibromatous hyperplasia." The operative result was good. The patient could retain urine for 2 to 3 hours. He could sense a warning that urination was about to take place. He voided as much as 250 cubic centimeters of urine each time. The bladder was emptied. This patient received additional therapy in the Section on Physical Medicine, and at the time of his dismissal, with the use of braces and crutches, he could get about alone, using a swinging gait. He could put on his braces, get up from floor alone, climb stairs, and get in and out of an automobile.

Ninth illustrative case. The case represents a transverse lesion of the spinal cord secondary to surgical removal of a tumor of the spinal cord. The patient was ambulatory. Marked hydronephrosis was present. A male 17 years old was admitted to the clinic on September 11, 1945. In August, 1944, operation had been performed elsewhere for teratoma of the spinal cord, and complete removal of the tumor had been effected. After the operation saddle anesthesia, weakness of the lower extremities and urinary retention developed. A retention catheter had been inserted and kept in place for 10 days. Some degree of sensation had then returned to the bladder, but the patient had been unable to void. "Leakage" of urine was constant. The patient tried to control it with a penile clamp. Residual urine amounted to 1,200 cubic centimeters. The patient had been catheterizing himself 2 times daily. Bilateral lumbar pain was relieved by urethral catheterization. Neurologic examination disclosed an intraspinal lesion, with marked damage to motor roots. The sensory level was at the fourth or fifth lumbar vertebra. The abdominal muscles were normal. An excretory urogram was made; there was marked delay in visualization, but bilateral hydro-

nephrosis of grade 2 plus was observed. Cystoscopy showed trabeculation of grade 3. The detrusor urinae muscle was spastic, and spastic contraction of the vesical neck was marked. At transurethral resection carried out on September 20, 1945, 2 grams of tissue was removed. The pathologic report was "hypertrophied muscle." The operative result was that residual urine persisted. At transurethral resection on September 28, 1945, 4 grams of tissue was removed. The pathologic report was "adenofibromatous hyperplasia." The result of this operation was that residual urine persisted. An episode of acute pyelonephritis occurred. At transurethral resection on October 15, 1945, 5 grams of tissue was removed. The pathologic report was "adenofibromatous hyperplasia." The operative result was good. The patient emptied the bladder completely, and voided every 2 to 3 hours. He strained with the abdominal muscles and employed manual compression. The stream was satisfactory. The patient could remain dry. An answer to a follow-up letter 3 months later indicated that the patient was well, and that the urine was clear.

Tenth illustrative case. This case represents transverse myelitis secondary to previous administration of a spinal anesthetic agent, with saddle anesthesia and vesical dysfunction. The patient, a man 22 years old, was admitted to the clinic on August 22, 1944. Sixteen months previous to admission, the patient had received a spinal anesthetic agent prior to performance of appendectomy. Since that time he had experienced marked difficulty with bowel movements and great difficulty in voiding. He required much straining and manual compression to eliminate urine, and residual urine amounted to more than 500 cubic centimeters. He used enemas and had to strain to evacuate the bowel. Neurologic examination revealed anesthesia in the saddle area, such as would be caused by a lesion of the spinal cord at the fourth or fifth sacral segment. There was no motor involvement. The abdominal muscles were normal. An excretory urogram disclosed nothing significant. Cystoscopy revealed mild trabeculation and slight contracture of the vesical neck. At transurethral resection on September 14, 1944, 4 grams of tissue was removed. The pathologic report was "adenofibromatous hyperplasia." The operative result was good. The patient emptied the bladder completely. He still had to use a moderate degree of abdominal straining to void.

Eleventh illustrative case. This case is representative of a war injury (bullet wound) in the thoracic portion of the spinal column, with no recovery of paralysis of extremities. A man 22 years old was admitted to the clinic on November 23, 1945. As a marine, he had been wounded during the action in the Gilbert Islands in November, 1943. He had been shot through the spinal column with a .45 caliber bullet from an automatic pistol. A suprapubic tube had been put in place 2 weeks after the patient's injury, and had been left in place for 8 months, after which it had been removed. The wound had been allowed to close. When we saw the patient he voided every 2 to 2½ hours. He had a sensation of fullness over the lower part of the abdomen before micturition would occur. He remained dry between urinations. Residual urine amounted to 400 cubic centimeters. Neurologic examination disclosed complete spastic paralysis of the lower extremities. The sensory level was at the eighth thoracic segment. The upper abdominal muscles exhibited loss of strength of grade 2 minus. Loss of strength in the lower abdominal muscles was grade 4 minus. Cystoscopy disclosed trabeculation of grade 2 plus. The detrusor urinae muscle was spastic. At transurethral resection, carried out on December 10, 1945, 4 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." The operative result was good. The patient voided every 2 hours; the amount of residual urine was less than 50 cubic centimeters. The patient sensed a warning when he was ready to void. He was able to remain dry.

Twelfth illustrative case. The case is representative of a traumatic lesion of the fifth cervical segment of the spinal column, with quadriplegia; there was partial recovery of the arms but no recovery of the lower extremities. A man 25 years old was admitted to the clinic on October 23, 1945. In September of 1936 he had sustained fracture of the sixth cervical vertebra in an automobile accident. Quadriplegiahad occurred immediately. Laminectomy had been performed 7 days later. Some degree of recovery in arms had followed, but spastic contracture of the hands had persisted. The patient had been unable to feed himself. An indwelling catheter had been employed for several months after the injury. At the time we saw the patient he was wearing a urinal constantly. The bladder expelled its contents about every hour. Very foul, thick residual urine amounted to 120 cubic centimeters. The patient experienced a feeling of flushing and perspiration before voiding occurred. Neurologic examination disclosed spastic paralysis of the lower extremities, but partial use of the arms remained. The sensory level was at the fifth cervical segment. The abdominal muscles were completely paralyzed. An excretory urogram disclosed a stone in the upper third of the right ureter, and no function of the right kidney. The left kidney was normal. Cystoscopy revealed the bladder to be trabeculated to grade 3 plus. Marked hypertrophy of the wall of the bladder was noted. At transurethral resection, carried out on October 29, 1945, 7 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." After the operation residual urine amounted to 400 cubic centimeters. At transurethral resection, carried out on November 14, 1945, 6 grams of tissue was removed. The pathologists reported "inflamed muscle." The result was that residual urine amounting to 300 cubic centimeters persisted. When transurethral resection was performed for the third time on January 9, 1946, 3 grams of tissue was removed. The pathologists reported "granulomatous prostatitis." Transurethral resection was

carried out for the fourth time on January 23, 1946, during which 4 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." Patient is still under observation at the time of writing. He voids every hour. Much perspiring occurs before evacuation of urine. The amount of residual urine now has been reduced to 200 cubic centimeters and seems to be diminishing gradually.

In evaluating the results of transurethral resection for cord bladder, the surgeon must remember to allow the patient sufficient time for recovery. It must be remembered that the treatment of this condition is not directed toward eradication of the underlying process that is producing the disturbance, but rather, toward correction of a mechanical defect and teaching the patient to substitute other muscles and other muscular activities to achieve complete vesical evacuation and urinary "continence." In respect to this statement, it must be made clear that the abdominal muscles and diaphragm are very important. If the abdominal muscles are intact, the response of the patient to transurethral resection will be more rapid and dramatic than would be the case otherwise. He is instructed to void at certain intervals (unless he experiences some warning of impending micturition). When urination commences, he sets his diaphragm, so to speak, strains with his abdominal muscles and, if necessary, employs manual compression of the area over the bladder. In this way complete emptying of the bladder is possible. Where the more efficient type of cord bladder is present, such a degree of abdominal straining may not be necessary. It is of paramount importance that the bladder be emptied *completely*, or as nearly completely as possible, at each voiding, to enable the patient to stay dry and to avoid frequent discharges of urine.

If the lesion is situated sufficiently high in the cord, so that the abdominal muscles are not under voluntary control, the problem may be more difficult and manual expression may be of greater importance. Injuries in the higher thoracic segments of the spinal cord, however, often result in a more efficient "automatic" type bladder, a condition which may more than compensate for the loss of function of the abdominal musculature.

As stated previously, because of the many and varied problems arising in the care of these patients sufficient time must be given them to recover before the results of treatment are evaluated. The experience of most transurethral surgeons is such that in the average instance of urinary retention arising from benign prostatic hyperplasia, if the operation has been complete, normal micturition is expected to occur almost immediately after removal of the urethral catheter. In many cases of cord bladder just such an excellent immediate response is achieved, but in others several days or weeks of observation and care are required before a completely satisfactory result is forthcoming. The surgeon should not become discouraged when a patient responds slowly, and should not hesitate to perform transurethral resection several times, if necessary, before he considers his treatment a failure. Some patients are very co-operative, and quickly learn how to make the most of their remaining normal muscles and available bodily functions. Others, however, are depressed and must be taught slowly.

World War II has given much impetus to study of the general problem of the patient who has an injury to the spinal cord. It has been demonstrated by the medical services of the armed forces that, with the co-operation of the neurologist, neurosurgeon, orthopedist, and physiotherapist, most patients thus afflicted need not be confined to bed for the rest of their lives. They may be rehabilitated, and become self-sufficient and useful members of society (4, 10). By means of leg braces and crutches, they learn how to become ambulatory with a tripod or swinging gait. Disabling spastic contractures are cared for surgically. By virtue of the fact that the patient is up and about, the formation of urinary stones, in most cases, is prevented.

The chief obstacle to rehabilitation of the average patient who has an injury to the spinal cord has been vesical dysfunction. This situation has stimulated urologists, both in the armed forces and in civilian life, to find better means of treating this condition. Thompson (18) and others (17, 20) have demonstrated that a large percentage of these patients can be made to empty their bladders completely and to remain continent and dry by means of transurethral resection. We are

anxiously awaiting complete reports of their experience.

Data concerning 15 patients who had multiple sclerosis and miscellaneous lesions of the spinal cord. When collecting cases for a study of this kind, the urologic surgeon is always confronted with the problem of discarding numerous cases in which resection was performed for an obstructive vesical neck or prostate gland, yet in which some kind of disease of the spinal cord was also present. It is obvious that in such cases there is not always a sharp line of demarcation between a "cord or neurogenic bladder" and "prostatic obstruction"; often there is a combination of both lesions. In collecting data concerning this group of 15 patients who had miscellaneous lesions, we chose them because the neurologic lesion seemed by far to be the predominating factor in the vesical dysfunction.

Multiple sclerosis.—Vesical dysfunction is a condition not uncommonly complained of by patients suffering from multiple sclerosis. Although the severity of the vesical symptoms tends to wax and wane as the severity or characteristics of the disease change, the type of vesical dysfunction remains more or less constant. Vesical symptoms usually consist of urgency and frequency of micturition, unsatisfactory urinary stream, difficulty in initiating and maintaining the urinary stream and, at times, urgency incontinence. Most of the patients have substantial amounts of residual urine. The usual complications of retention of urine, such as vesical calculi and urinary infection, may be present.

In this total series of 45 patients there were, as said previously, 7 patients who had multiple sclerosis for which transurethral resection was performed. Four of these 7 patients were less than 45 years old. One of the patients was a woman. Although most such patients can be helped considerably and the residual urine eliminated by performance of transurethral resection, results of this operation have not been consistently brilliant. Only 3 of the 7 patients obtained a "good" result, and 4, although their condition was definitely improved, did not obtain perfect recovery. All of the 4 patients who achieved only "fair" results underwent transurethral resection only once each. It may be that if resection had been carried out a second time in each case, a more nearly perfect result might have been achieved. Our experience in this group of cases is still too limited to warrant the making of any far-reaching conclusions. It can be said, however, that the results to date are sufficiently encouraging to justify the statement that the urologic surgeon should not hesitate to advise transurethral resection in these cases. The thirteenth and fourteenth illustrative cases may be of help in an understanding of this condition.

Thirteenth illustrative case. This case is representative of vesical dysfunction associated with multiple sclerosis, and relief achieved by transurethral resection. The patient, a man 41 years old, was admitted to the clinic on February 1, 1945. He gave a history typical of multiple sclerosis of several years' duration, with ataxia, poor vision, and so forth. The neurologic diagnosis of multiple sclerosis with retrobulbar neuritis had been made. In the 2 years prior to the patient's admission difficulty in urination had developed; it consisted of hesitancy, small urinary stream, difficulty in maintenance of stream, frequency of micturition, and nocturia requiring voiding 2 to 3 times. Urologic examination disclosed 450 cubic centimeters of residual urine. Cystoscopic examination revealed the bladder to be trabeculated to grade 2. Slight trilobar prostatic hyperplasia of grade 1 was noted. At transurethral resection performed on February 28, 1945, 6 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." The operative result was good. When the patient was dismissed he was voiding well, and had no residual urine. He was readmitted on October 3, 1945. He was still voiding well, but catheterization disclosed residual urine amounting to 350 cubic centimeters. At transurethral resection performed on October 19, 1945, 7 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." The operative result was good. The urinary stream was satisfactory. Residual urine amounted to less than 50 cubic centimeters. There was no frequency of micturition.

Fourteenth illustrative case. This case is representative of vesical dysfunction associated with multiple sclerosis and incomplete relief afforded by transurethral resection. The patient, a man 39 years old, was admitted to the clinic on January 24, 1945. His history was typical of multiple sclerosis, with ataxic gait, weakness of the left leg, nystagmus, and tremor of the head and tongue. The neurologic diagnosis of multiple sclerosis was made. Urinary symptoms consisted of frequency and urgency of micturition, dribbling, and nocturia. At times the patient had suffered from urgency incontinence of urination. Urologic examination revealed residual urine amounting to 120 cubic centimeters. Cysto-

scopy showed the bladder to be trabeculated to grade 1 plus. A small collar of tissue surrounded the vesical neck. At transurethral resection, performed on February 5, 1945, 7 grams of tissue was removed. The pathologists reported "adenofibromatous hyperplasia." The operative result was fair. Some degree of urgency of urination persisted. At the time of dismissal the patient voided about every hour and a half. Residual urine amounted to about 75 cubic centimeters. (Note: Patient did not undergo cystoscopy again. A second performance of transurethral resection may have been indicated.)

Miscellaneous lesions of the spinal cord.—The 8 remaining cases involving miscellaneous lesions of the spinal cord are of only passing interest. One patient was a man 32 years old who had severe diabetes mellitus associated with marked diabetic retinitis. For several months he had had about 500 cubic centimeters of infected residual urine; this condition had been ascribed to "diabetic neuritis." This, of course, was a difficult diagnosis to prove. Transurethral resection was done in two stages. A total of 8 grams of tissue was removed; this allowed the patient to empty his bladder completely. Normal micturition was restored. The pathologic report on the tissue removed was "adenofibromatous hyperplasia."

Four cases of combined sclerosis are of interest in that 3 of the 4 patients secured good results from the operation. This outcome would tend to dispel the fear of prostatic surgery which has always been entertained by the urologists and neurologists in respect to such patients. This would apply regardless of whether the physician believes that the vesical dysfunction is primarily neurologic or prostatic in origin.

The remaining 3 cases are representative of some of the obscure neurologic conditions which are found associated with vesical dysfunction. Two of the patients had undergone surgical exploration of the spinal cord, and the unsatisfactory diagnosis of "arachnoiditis" subsequently had been made. The condition of 1 patient had been diagnosed as "degenerative lesion of the spinal cord." In 2 of these 3 cases the result of resection was unsatisfactory.

CONCLUSIONS

Cord bladder no longer is a hopeless situation for which nothing can be done. Although knowledge of the neurophysiologic aspects of micturition is still too imperfect to permit institution of therapy based on correction of the underlying causative process, satisfactory vesical function can be restored by mechanical adjustment of the vesical neck and by teaching the patient to use unaffected muscles to aid him to void. Mechanical adjustment of the vesical neck is brought about by transurethral resection.

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APPENDICITIS IN CLEVELAND

Final Report

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IN previous publications (1, 2, 3, 4, 5, 6, 7) preliminary summaries of the work of the Cleveland Appendicitis Survey Committee have been presented. After much delay due to the War, this summarized report is finally here published. One of us (H. W. G.) has gone to great lengths to make very extensive statistical analyses of the findings of the Committee which are available, as noted, elsewhere.[1]

THE SURVEY

The work of collecting statistics was begun in 1939 and finished in 1942. We attempted to study the subject of appendicitis, acute, ruptured with peritonitis and with abscess, by collecting the records of all the cases in nearly all the Greater Cleveland hospitals for a 12 year period, from January 1, 1930, to January 1, 1942. Our grand total became 19,399 case records. For diagnostic criteria we relied on the hospital pathologists' reports and carefully avoided any questionable cases of "chronic," "subacute," or "interval" appendicitis. In other words, these 19,399 cases represent, at the mildest, genuine acute appendicitis, or worse, suppurative with rupture, and rupture with abscess. Further, we avoided case records of patients who had some other acute disease as well as acute appendicitis.

In the text to follow, simple acute appendicitis is termed "unruptured"; suppurative with peritonitis and rupture is called "peritonitis" and ruptured with abscess, simply "abscess."

The object of this study was to determine: (1) the best procedure in the hospital relative to anesthetic, incision, and drainage; and (2) the effect of various factors on recovery: sex, age, delay, laxative, condition of appendix, anesthetic, incision, drainage, surgeon, hospital, year, economic status.

APPENDICITIS IN CLEVELAND

An analysis of the 19,399 operations for acute appendicitis performed in 16 hospitals in the Cleveland area during the 12 years, 1930 to 1942, showed 4.8 deaths per 100 operations.

The crude fatality rates varied by age, sex, delay, laxative, condition of appendix, anesthetic, incision, drainage, surgeon, hospital, and the year as shown on Table I and Charts 1 to 6 which summarize the results of the detailed tabulations which are included in the full report.

Some factors were found to exert great influence, while others exerted little or no influence on the fatality rate. The more important factors have been examined singly uninfluenced by the others. The effect that each exerted upon the fatality rate has been given particular attention.

Condition of appendix, drainage, delay, age. The factors which exerted the greatest effect upon satisfactory recovery were condition of appendix, drainage, delay, and age. They had many more times the effect upon the crude fatality rate than did their nearest competitors.

The factor which showed the greatest variation was condition at time of operation. Most persons had an unruptured appendix, 75 per cent of the 19,399. Those with unruptured appendices suffered a crude fatality

In preparing this report Mr. Green, director of Statistics and Research, Cleveland Health Council, and Dr. Watkins, Chairman Survey Committee, were assisted by Doctors D. C. Darrah, R. M. Hosler, H. H. Pevaroff, J. D. Brett, J. H. Lazzari, C. W. Rotter, S. J. Restifo, H. R. Hathaway, W. A. Boukalik, John Kelker, J. F. Renshaw, A. F. Sydow, B. B. Larsen, J. M. Rossen, John Budd, S. L. Feldman, R. J. McNamee, P. L. Suhay, George Crile, and Fred Kelly, members of the Appendicitis Survey Committee.

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[1]The extensive detailed report complete with tables, maps and charts, which cannot be reproduced here because of lack of space, is available upon request to the authors.

TABLE I.—APPENDICITIS OPERATIONS IN 16 HOSPITALS IN THE CLEVELAND AREA, 1930–1941

	Total	Per cent	Recovered	Died	Unknown	Crude fatality rate—S.E.
Sex Total	19,399	100.0	18,443	936	20	4.83± .15
Male	11,236	58.0	10,674	553	9	4.93± .20
Female	8,153	42.0	7,760	383	10	4.70± .23
Unknown	10	-	9	—	1	–
Age Total	19,399	100.0	18,443	936	20	4.83± .15
Under 5	302	1.6	250	52	–	17.22±2.17
5–9	1,373	7.1	1,287	85	1	6.20±.65
10–14	2,663	13.8	2,588	73	2	2.74± .32
15–19	3,189	16.4	3,128	59	2	1.85± .24
20–24	3,029	15.6	2,969	56	4	1.85± .24
25–29	2,250	11.6	2,202	45	3	2.00± .30
30–34	1,682	8.7	1,617	63	2	3.75± .46
35–39	1,289	6.7	1,227	59	3	4.59± .58
40–44	1,018	5.3	951	66	1	6.49± .77
45–49	876	4.5	791	84	1	9.60±1.00
50–54	631	3.3	565	66	–	10.46±1.22
55–59	467	2.4	392	74	1	15.88±1.73
60–64	300	1.5	243	57	–	19.00±2.26
65–69	154	.8	109	45	–	29.22±3.66
70	136	.7	87	49	–	36.03±4.15
Unknown	40	–	37	3	–	7.50±
Delay in days Total	19,399	100.0	18,443	936	20	4.83± .15
Under 1	3,522	18.7	3,486	34	2	.97± .50
1.0–1.4	5,938	31.6	5,800	134	4	2.26± .19
1.5–1.9	2,233	11.9	2,148	82	3	3.68± .40
2.0–2.4	2,774	14.8	2,608	164	2	5.92± .45
2.5–2.9	505	2.7	457	47	1	9.33±1.30
3.0–3.9	1,673	8.9	1,495	177	1	10.59± .75
4.0–4.9	717	3.8	638	78	1	10.89±1.16
5	1,430	7.6	1,235	192	3	13.45± .90
Unknown	607	–	576	28	3	4.64±
Laxative Total	19,399	100.0	18,443	936	20	4.83± .15
Laxative	4,713	35.6	4,334	376	3	7.98± .30
No laxative	8,533	64.4	8,214	308	11	3.61± .20
Unknown	6,153	–	5,895	252	6	4.10±
Condition Total	19,399	100.0	18,443	936	20	4.83± .15
Unruptured	14,605	75.4	14,453	137	15	.94± .08
Peritonitis	3,216	16.6	2,613	600	3	18.67± .69
Abscess	1,550	8.0	1,353	196	1	12.65± .84
Unknown	28	–	24	3	1	11.11±

	Total	Per cent	Recovered	Died	Unknown	Crude fatality rate—S.E.
Anesthetic Total	19,399	100.0	18,443	936	20	4.83± .15
General	16,973	87.8	16,196	763	14	4.50± .16
Spinal	2,235	11.6	2,100	133	2	5.96± .50
Local	126	.6	98	28	–	22.22±3.70
Unknown	65	–	49	12	4	19.67±
Incision Total	19,399	100.0	18,443	936	20	4.83± .15
McBurney	11,538	61.2	11,091	443	4	3.84± .17
No McBurney	7,317	38.8	6,849	455	13	6.23± .23
Unknown	544	–	503	38	3	7.02±
Drainage Total	19,399	100.0	18,443	936	20	4.81± .15
Drain	6,634	34.5	5,812	813	9	12.27± .40
No drain	12,577	65.5	12,457	111	9	.88± .08
Unknown	188	–	174	12	2	6.45±
Surgeon Total	19,399	100.0	18,443	936	20	4.83± .15
Staff	4,624	23.9	4,322	297	5	6.43± .36
Private	14,759	76.1	14,118	635	6	4.30± .16
Unknown	16	–	3	4	9	57.14±
Hospital Total	19,399	100.0	18,443	936	20	4.83± .15
C	1,579	8.1	1,532	47	–	2.98± .43
K	1,164	6.0	1,123	40	1	3.44± .53
N	1,685	8.7	1,617	60	8	3.58± .45
E	241	1.3	232	9	—	3.73±1.22
P	2,216	11.4	2,134	81	1	3.66± .40
I	858	4.4	822	35	1	4.08± .68
B	2,287	11.8	2,183	102	2	4.46± .43
G	651	3.3	620	29	2	4.47± .81
J	1,508	7.8	1,433	74	1	4.91± .56
F	522	2.7	495	27	–	5.17± .97
M	626	3.2	592	34	–	5.43± .91
O	1,714	8.8	1,615	97	2	5.67± .56
H	1,485	7.7	1,392	93	–	6.26± .63
L	770	4.0	719	51	–	6.62± .90
A	1,614	8.3	1,496	117	1	7.25± .40
D	479	2.5	438	40	1	8.37±1.26
Year Total	19,399	100.0	18,443	936	20	4.83± .15
1930	1,438	7.4	1,337	97	4	6.76± .66
1931	1,457	7.5	1,358	96	3	6.60± .65
1932	1,418	7.3	1,321	94	3	6.64± .66

TABLE I.—APPENDICITIS OPERATIONS IN 16 HOSPITALS IN THE CLEVELAND AREA, 1930–1941—Continued

	Total	Per cent	Recovered	Died	Unknown	Crude fatality rate—S.E.		Total	Per cent	Recovered	Died	Unknown	Crude fatality rate—S.E.
Year							Year						
1933	1,412	7.3	1,332	80	–	5.67± .62	1938	1,727	8.9	1,661	64	2	3.71± .46
1934	1,596	8.2	1,503	92	1	5.77± .58	1939	1,771	9.1	1,702	66	3	3.73± .45
1935	1,554	8.0	1,464	90	–	5.79± .59	1940	1,676	8.7	1,632	44	–	2.63± .39
1936	1,646	8.5	1,572	73	1	4.44± .51	1941	1,938	10.0	1,881	56	1	2.89± .38
1937	1,766	9.1	1,680	84	2	4.76± .51							

NOTE:—Fatality rates when adjusted for age, delay, condition, anesthetic, and drain were quite different from these crude fatality rates. S.E. indicates standard error.

rate of 0.9, while those with appendix ruptured with peritonitis suffered a crude rate of 18.7, that is, a rate 21 times as great. Even when adjusted for age, delay, drainage, and anesthetic, these differences were still found to be great.

The next most important factor was drainage. In cases of unruptured appendix with low fatality rates usually drainage is not carried out while in those of ruptured appendix with peritonitis with high fatality rates drainage usually is done. In those with no drain the crude rate was 0.9 per cent, while in those with drainage the crude rate was 12.3, or 14 times as high as the no drain cases. When adjusted for condition, age, delay, and anesthetic, the differences were not nearly as great.

Delay ranked next. Those patients who were operated upon within 24 hours of the time of the appearance of the first symptoms experienced a crude fatality rate of 1.0 compared with those who delayed 5 or more days with a crude fatality rate of 13.5 or 13 times that of the former delay group. When adjusted for age, drainage, anesthetic, and condition of appendix, these differences were somewhat smaller.

Age ranked high, and depending upon what age groups were compared, ranked first, second, third, or lower. Few young children were operated upon for appendicitis, many older children and young people, and few persons of older ages. The fatality rate was high at very young ages, low for those in their teens and twenties, and high for older persons. Those of 20 to 24 years of age had a crude rate of 1.9, while those 50 years and over had a crude rate of 17.2 or 9 times that of the former group. When adjusted for delay, drainage, anesthetic, and condition of appendix, these differences were still very large.

Hospital, year, laxative, economic status. The factors of lesser importance fell in the following order: hospital, year of operation, laxative, and economic status. The effect of each of these factors was difficult to determine.

The crude fatality rates varied from 3.0 in one hospital to 8.4 in another. If adjusted for age, delay, condition, anesthetic and drainage, much smaller differences would probably have been evidenced.

The crude fatality rate of 6.8 in 1930 decreased to 2.6 in 1940 and 2.9 in 1941. This decrease was due to less delay, more favorable condition of appendix at the time of operation, and the changes in anesthetic, and drainage.

Persons taking laxatives or enemas prior to operation suffered higher crude fatality rates than did those having no laxatives. Those taking laxatives or enemas showed a crude rate of 8.0 contrasted with those with no laxatives with a crude rate of 3.6. These rates when adjusted for condition, anesthetic, drainage, age, and delay were not significantly different. However, in patients taking laxatives or enemas rupture is more frequent than in those not taking laxatives. Thus laxatives exert indirectly an unfavorable effect upon the fatality rates.

Incision, surgeon, anesthetic, sex. The factors of least importance were type of incision, surgeon, anesthetic, and sex.

Those with McBurney incision showed a crude fatality rate of 3.8 and those with no McBurney 6.2 or 63 per cent higher. When

TABLE II.—MAXIMUM VARIATIONS IN CRUDE FATALITY RATES

Minimum					Maximum				
Factor	Condition	Number of		Crude fatality rate	Condition	Number of		Crude fatality rate	Per cent maximum is of minimum
		Cases	Deaths			Cases	Deaths		
Condition	Unruptured	14,605	137	.9	Peritonitis	3,216	600	18.7	2,078
Drainage	No drainage	12,577	111	.9	Drainage	6,634	813	12.3	1,367
Delay	Under 1 day	3,522	34	1.0	5+ days	1,430	192	13.5	1,350
Age	20–24 years	3,029	56	1.9	50+ years	1,688	291	17.2	905
Hospital	C	1,579	47	3.0	D	479	40	8.4	280
Year	1940	1,676	44	2.6	1930	1,438	97	6.8	262
Laxative	No laxative	8,533	308	3.6	Laxative	4,713	376	8.0	222
Economic status	3 highest	934	18	1.9	3 lowest	806	33	4.1	216
Incision	McBurney	11,538	443	3.8	No McBurney	7,317	455	6.2	163
Surgeon	Private	14,759	635	4.3	Staff	4,624	297	6.4	149
Anesthetic	General	16,973	763	4.5	Spinal	2,235	133	6.0	133
Sex	Female	8,153	383	4.7	Male	11,236	553	4.9	104

adjusted for age, delay, drainage, anesthetic, and condition of appendix, only slight differences were evident by incision except for the ruptured with peritonitis group with drainage and general anesthetic.

The crude fatality rate varied from 4.3 for the private surgeons' cases to 6.4 for staff cases or 49 per cent higher. When adjusted for age, delay, drainage, anesthetic, and condition of appendix, no difference was evident.

Most persons are given a general anesthetic for an appendix operation. The crude fatality rate varied from 4.5 for patients taking a general anesthetic to 6.0 for those taking a spinal anesthetic or 33 per cent higher. When adjusted for age, delay, condition of appendix, and drainage, the relationship was quite different—lower fatality rates being shown for spinal anesthetic than for general.

TABLE III.—CRUDE FATALITY RATE BY CONDITION AND DRAINAGE

Condition	Drainage	Cases	Deaths	Crude fatality rate
Total	Drainage	6,156	750	12.18± .42
	No drainage	11,824	105	.89± .02
Unruptured	Drainage	2,123	58	2.73± .35
	No drainage	11,409	65	.57± .07
Ruptured	Drainage	4,033	692	17.16± .59
	No drainage	415	40	9.64±1.45

Females experienced a crude rate of 4.7 and males 4.9, 4 per cent higher. If adjusted by delay, drainage, condition of appendix, and anesthetic, no significant difference would have been evident.

TRUE DIFFERENCES IN FATALITY RATES

Some of the extreme differences shown on the preceding pages, were real while others were complicated by the combined effects exerted by various factors.

For example, drainage showed a crude fatality rate 13.9 times as high as did no drainage, but since most unruptured cases with low crude fatality rates have no drainage, and most ruptured cases with high crude fatality rate have drainage, the variation in crude fatality rates of cases by drainage, unless more closely examined, is very misleading.

In order to measure the effect of the various factors, it is necessary to compare groups in which all factors are the same except the one being examined. It is possible to do this completely only in laboratory experiments. With even as many as 19,399 appendix operations, small numbers complicate the procedure when the group is broken into its simplest elements.

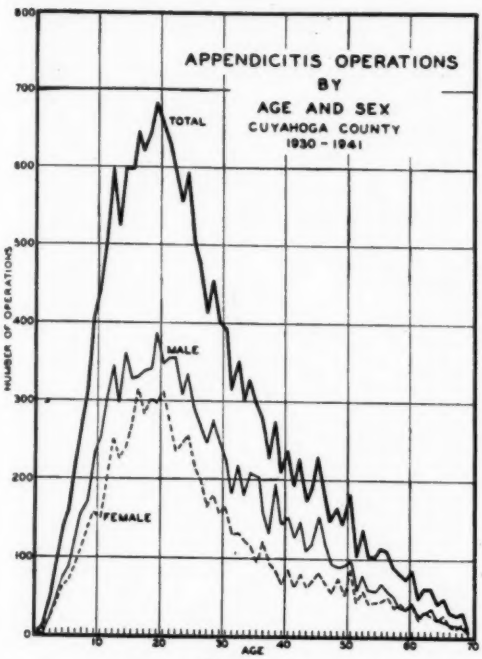

Chart 1.

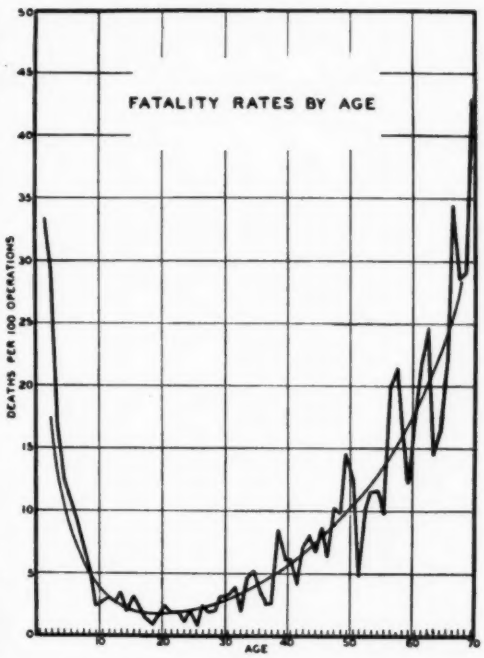

Chart 2.

It has accordingly been necessary to assume that laxative, hospital, year, and economic status are constant and exert the same influence on each group. The cases and deaths were tabulated by age and delay for each of 72 groups.[1]

The first pair consisted of patients with appendix unruptured, no drainage, general anesthetic, McBurney incision, and private surgeon and patients with appendix unruptured, no drainage, general anesthetic, McBurney incision, and staff surgeon.

When the fatality rates for this or any other pair differing by but one factor are adjusted to a standard age and delay distribution, the true difference is shown.

Comparing these 72 groups in pairs with identical factors except the one under examination, the statistical significance of each factor is determined.

Differences caused largely by age and delay. Differences in crude fatality rates between comparable groups were caused largely by differences in age and delay. To illustrate this point the two largest groups are presented. These two groups were unruptured private type cases with general anesthetic and no drainage, with no McBurney incision and with McBurney incision, respectively.

The crude fatality rate of 3,011 cases with no McBurney incision of $.70 \pm .15$ was 30 per cent higher than was the crude fatality rate of the 4,660 cases with McBurney incision of $.49 \pm .10$.

Those with McBurney incision were younger with lower fatality rates.

Furthermore, the McBurney incision group had not delayed operation for as long a period, and this factor also gave it lower fatality rates.

When age-delay specific fatality rates of each of these groups were applied to the distribution of cases by age and delay of the two groups taken together, the adjusted fatality rates differed very slightly: McBurney incision, $.52 \pm .11$; no McBurney incision, $.57 \pm .14$. Thus, when the fatality rate of Mc-

[1]Tables reproduced in the full report.

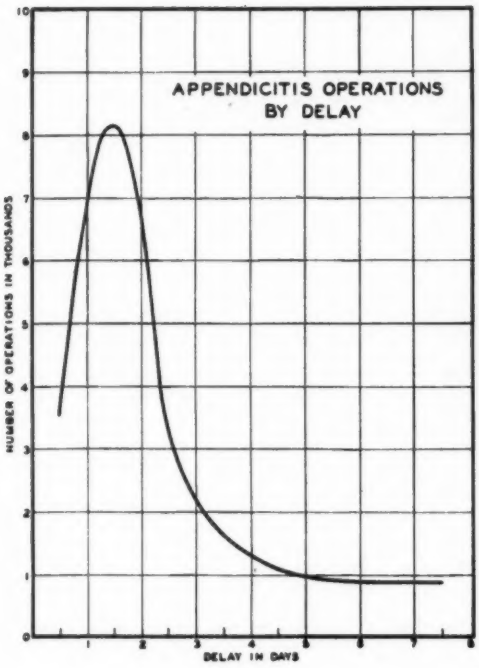

Chart 3.

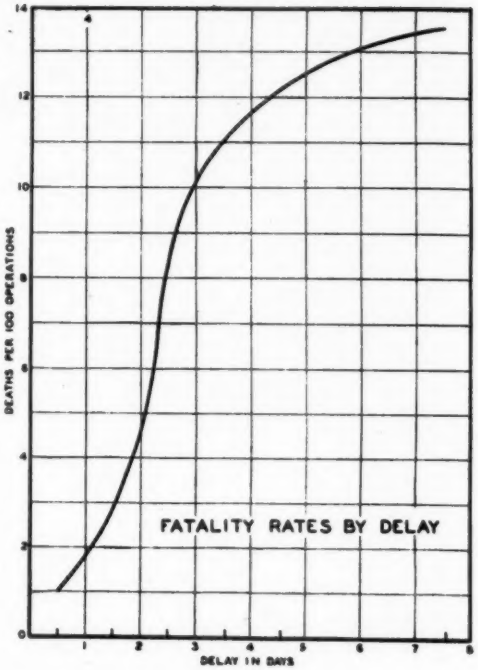

Chart 4.

Burney incision cases was compared with that of the no McBurney incision cases adjusted for all measured factors, no significant differences in rates were evident.

SUMMARIZED TRUE RATES

Crude fatality rates were further adjusted in order to find the true fatality rates. The results of this procedure, presented in detail in the full report but omitted for lack of space here, are summarized:

Incision

In ruptured cases with peritonitis, with general anesthetic and drainage, the fatality rate with no McBurney incision (21.27) is 1.3 times that with McBurney incision (16.31).

While other comparable groups showed no significant differences, the adjusted fatality rates for those with no McBurney incision were higher in every instance than were those with McBurney incision.

Anesthetic

In ruptured cases with peritonitis and drainage, the rate with general anesthetic (19.09) is 1.6 times spinal (12.26).

Drainage

With unruptured appendix and general anesthetic, the rate if drained (1.56) is 2.6 times that if not drained (0.59).

With peritonitis and general anesthetic, if drainage can be avoided the rate (10.10) is much more favorable than with drains (18.54).

Condition of Appendix

With general anesthetic and no drainage, the rate in peritonitis cases (7.03) is 11.2 times that of unruptured (0.63).

With general anesthetic and drainage, the rate in peritonitis (17.82) is 5.7 times that of unruptured (3.11).

With general anesthetic and drainage, the rate in peritonitis (19.20) is 1.8 times that of abscess (10.66).

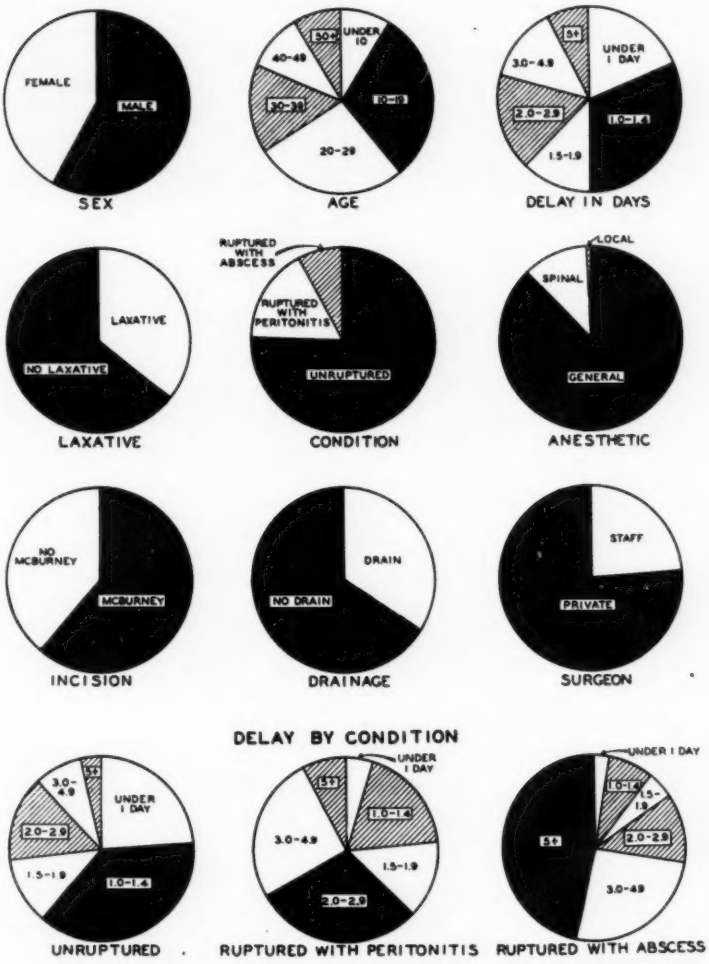

Chart 5. Appendicitis operations in 16 hospitals in Cleveland Area, 1930-1941.

With general anesthetic and drainage, the rate in abscess (10.41) is 2.6 times that of unruptured (3.98).

With spinal anesthetic and drainage, the rate in peritonitis (21.22) is 1.7 times that of abscess (12.16).

Delay

In the unruptured group with general anesthetic and no drainage the rate for those who delay 5 days or more (2.34) is 8.1 times that of those who delay less than 1.5 days (0.29).

In peritonitis with general anesthetic and drainage, the rate for those who delay 5 days or more (32.53) is 2.0 times that of those who delay less than 1.5 days (16.20).

Age

For unruptured cases with general anesthetic and no drainage, the rate for persons 50 years and over and under 5 years of age

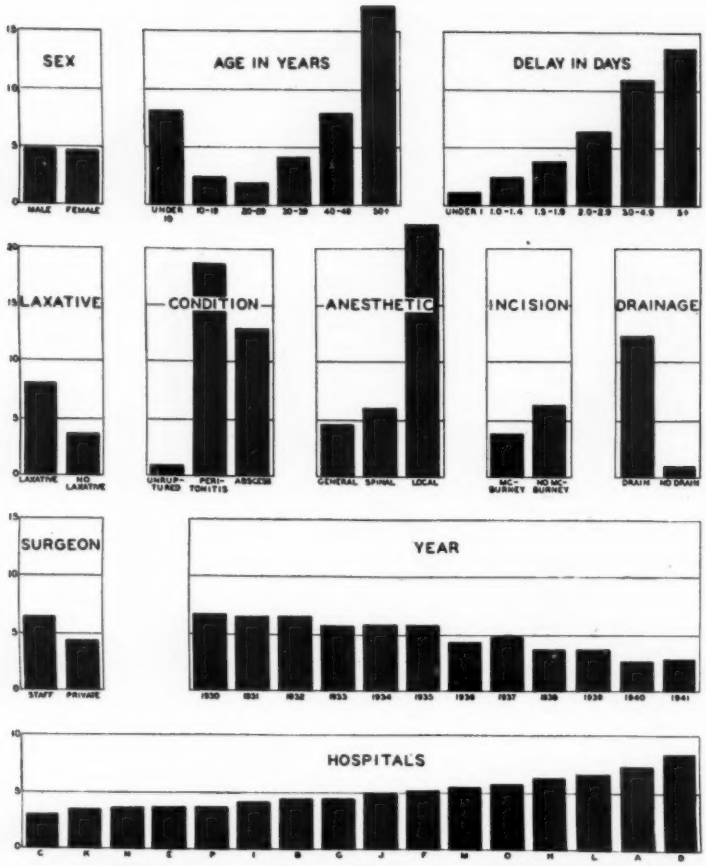

Chart 6. Fatality rates in appendicitis operations in 16 hospitals in Cleveland Area, 1930-1941.

(3.06) is 11.8 times that for the 5 to 29 year age group (0.26).

In peritonitis with general anesthetic and drainage, the rate for those 50 years and over and under 5 (32.27) is 2.3 times that of the 5 to 29 age group (14.34).

In abscess with general anesthetic and drainage, the rate for those 50 years and over and under 5 (20.26) is 3.1 times the 5 to 29 group (6.44).

Laxative

The fatality rate for no laxative cases compared with laxative cases adjusted by condition of appendix, anesthetic, drainage, age, and delay showed no significant difference, although in nearly all cases it was found that the adjusted fatality rate for the no laxative group was lower than that for the laxative group.

However, when adjusted for all factors except condition of appendix, no laxative cases had distinctly lower fatality rates than did laxative cases. This difference is accounted for by the fact that rupture less commonly accompanies the no laxative than the laxative cases.

Thirty-eight per cent of the laxative cases were ruptured at the time of operation, while 19 per cent of the no laxative cases were rup-

TABLE IV:—CASES, DEATHS, AND CRUDE FATALITY RATES BY LAXATIVE

Condition	With laxative				With no laxative			
	Cases	Per cent	Deaths	Crude fatality rate	Cases	Per cent	Deaths	Crude fatality rate
Unruptured	2,793	61.5	36	1.29 ± .21	6,361	81.3	42	.66 ± .10
Ruptured	1,747	38.5	313	17.92 ± .92	1,463	18.7	233	15.93 ± .30
Total	4,540	100.0	349	7.69 ± .39	7,824	100.0	275	3.51 ± .21

tured. The crude fatality rates of ruptured cases were many times as great as for the unruptured cases.

When fatality rates for all laxative and no laxative cases regardless of condition were adjusted for the age and delay or both: the laxative group (6.34) was 1.50 times the no laxative group (4.24).

Larger percentages of young children and old persons than of the group 10 to 39 years of age took a laxative.

Larger percentages of persons delaying operation took a laxative. The longer the delay, the larger the percentage with laxative.

Larger percentages of persons taking laxative than of those not taking laxative were ruptured. The unruptured cases in all age and delay groups had very much lower fatality rates than did those ruptured with peritonitis or abscess.

Changes with the Years

The crude fatality rate decreased from 6.8 to 2.9 during the 12 year period, 1930 to 1942, and has probably continued to decrease. Most of the factors affecting the fatality rate have become more favorable.

Delay. Fifty per cent more delayed less than 1 day. Fifty per cent more unruptured and more ruptured with peritonitis delayed less than 1 day. Half as many delayed 5 days or more. Half as many unruptured and fewer ruptured with peritonitis delayed 5 or more days. Median hours of delay decreased from 44 to 34. Unruptured decreased by 3 hours from 35 to 32 hours and ruptured with peritonitis decreased 10 hours from 62 to 52 hours. Ruptured with abscess did not change. The delay is well over 100 hours in most years.

Condition of appendix. More are unruptured. Somewhat fewer are ruptured with peritonitis. Half as many are ruptured with abscess.

Anesthetic. Fewer are given general anesthetic. Four times as many are given spinal anesthetic. Fewer are given local anesthetic.

Drainage. Half as many had drains. A third as many unruptured and fewer ruptured with peritonitis had drains. More had no drainage. More unruptured, twice as many ruptured with peritonitis, and a few more ruptured with abscess had no drainage.

Charts 7 and 8 show these changes which doubtless have continued to take place since 1941.

EFFECT OF ECONOMIC STATUS

A total of 3,273 of the 3,614 operations recorded in 1940 and 1941 were distributed geographically on a map included in the complete report. This distribution was not greatly different from that shown for total population.

TABLE V.—POPULATION, OPERATIONS, AND NUMBER PER 1,000 POPULATION BY ECONOMIC TENTHS

Economic tenth	Population 1940	Number of cases	Cases per 1,000 population
Total	1,065,376	2,904	2.7
Lowest	110,040	230	2.1
2	110,623	285	2.6
3	111,849	295	2.6
4	111,393	292	2.6
5	111,026	280	2.5
6	105,939	310	2.9
7	104,931	271	2.6
8	98,536	272	2.8
9	94,387	304	3.2
Highest	106,752	365	3.4

NOTE:—Data on place of residence incomplete for one hospital—a hospital serving the higher economic tenths.

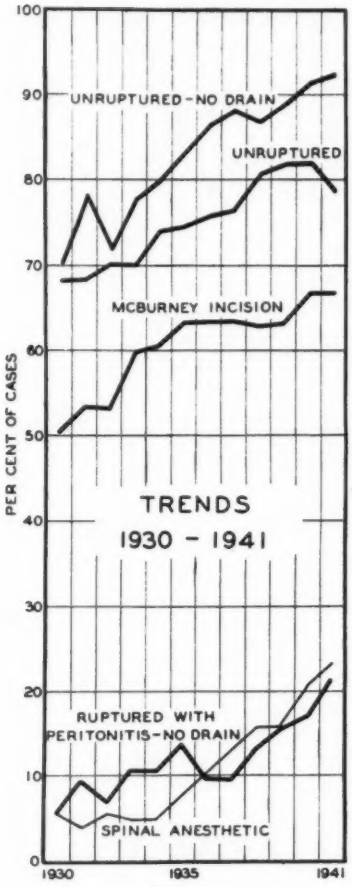

Chart 7.

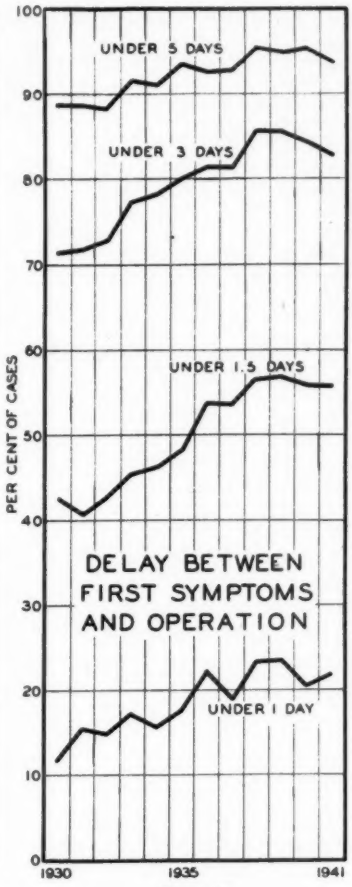

Chart 8.

The Cleveland five city area has, for statistical purposes, been divided into 10 economic tenths based on the average monthly rent of rented homes and rent equivalent of owned homes.

The population in April 1940 in each of these 10 areas, the number of operations, and the operations per 1,000 population are shown on Table V. This table shows slightly larger ratios in the higher economic tenths than in the lower economic tenths.

Slightly larger percentages of cases in the high economic tenths were unruptured than in the low economic tenths. The crude fatality rates were lower in the high economic tenths than in the low economic tenths except for those ruptured with abscess.

Larger percentages of the cases were staff type cases in the low economic tenths and smaller percentages in the high economic tenths.

Staff type cases show slightly more delay than private type cases and low economic tenth cases than high economic tenth cases. The median hours of delay for the group was 34.2, with 37.9 hours in the three low economic tenths and 32.9 in the three high economic tenths.

Higher percentages had general anesthetic in the high than in the low economic tenths.

The high crude fatality rates apparent in the low economic tenths and the low crude fatality rates in the high economic tenths might or might not have a significant difference if adjusted by age, delay, condition of appendix, anesthetic, and drainage.

Observations

It is evident that crude fatality rates are misleading when studying appendicitis operations. When rates are adjusted for age, delay, condition of appendix, anesthetic, and drainage, the true importance of each of the various factors becomes evident. This analysis of the results of 19,399 appendicitis operations allows certain observations to be made based on adjusted fatality rates.

1. Sex: There are no significant differences in the fatality rates.

2. Age: The younger the adult the lower the fatality rate. The 15 to 29 year age group has the lowest rate.

3. Delay: The less the delay, the lower the fatality rate.

4. Laxative: No laxative cases have slightly lower fatality rates. There are no significant differences in the fatality rate, except as laxative increases the proportion ruptured which condition shows rates much higher than unruptured.

5. Condition of the appendix: The unruptured appendix gives the more favorable fatality rate.

6. Anesthetic: Spinal anesthetic shows a lower fatality rate than general.

7. Incision: McBurney incision cases have slightly lower fatality rates.

8. Drainage: No drainage cases have a lower fatality rate than drainage cases under all conditions.

9. Surgeon: There is no significant difference between staff type and private type cases.

10. Hospital: There are too few cases at each hospital to determine the significance of the differences. Differences are probably largely due to differences in condition of appendix and delay and to the use of anesthetic and drainage.

11. Year: Probably no significant difference except as delay is shortened, and spinal anesthetic and no drainage become more common practice.

12. Economic status (available for 1940 and 1941 only): There are too few cases to determine the significance of the apparent differences.

Summary of True Differences

When all factors are kept constant except the pair under examination, it is possible to determine the true effect of each upon the fatality rate.

Condition of Appendix

Peritonitis: 11 times unruptured.
Peritonitis: 2 times abscess.
Abscess: 3 times unruptured.

Age

Unruptured: Over 50 and under 5 years, 12 times 5 to 29 years.
Peritonitis: Over 50 and under 5 years, 2 times 5 to 29 years.
Abscess: Over 50 and under 5 years, 3 times 5 to 29 years.

Delay

Unruptured: 5 days and over, 8 times under 1.5 days.
Peritonitis: 5 days and over, 2 times under 1.5 days.
Abscess: Makes no difference.

Drainage

Unruptured: Drainage 3 times no drainage.
Peritonitis: Drainage 2 times no drainage.
Abscess: Could not determine.

Anesthetic

Unruptured: General slightly higher than spinal.
Peritonitis: General 2 times spinal.
Abscess: General somewhat higher than spinal.

Laxative

Laxative slightly higher than no laxative.

McBurney Incision

No McBurney slightly higher than McBurney.

CONCLUSIONS

This study confirms present opinions of the effect on the fatality rates in appendicitis of age and sex distribution, delay in operation, condition of the appendix, and the use of laxatives. The work has revealed certain interesting points, some of them unique: Spinal anesthesia used in a large series has resulted in definitely lower fatality rates than general anesthesia; the patients operated on by the resident staffs of the hospitals had exactly as good chances of recovery as those operated on by private surgeons; there appears to be an advantage in using the McBurney incision instead of some other; the less favorable fatality rates indicate that drainage should not be routinely employed even in patients with peritonitis.

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ARTERIOVENOUS FISTULAS OF THE CERVICAL PORTION OF THE VERTEBRAL VESSELS

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ANEURYSMS and arteriovenous fistulas of the extracranial portion of the vertebral vessels are relatively rare. Matas in 1893 and Perrig in 1932 have contributed excellent reviews of traumatic lesions of the vertebral artery. Matas listed 20 patients with aneurysm of whom only 6 survived. Perrig was able to bring to 60 the number of extracranial vertebral aneurysms recorded in the literature. Nearly half of these patients died from the aneurysm or its treatment. Definite cures were obtained in 19 patients, improvement in 1, and the outcome was not recorded in 11. Heifetz has recently added a case of his own and collected 6 additional cases from the literature. Operative cures were apparently obtained in 5 of the 7 patients. Four cases from the American literature are included in these reviews, those of Matas, of Fenger, of Stone, and of Heifetz. The vast majority of the cases reported were arterial aneurysms and only a few were arteriovenous fistulas. When Callander reviewed 447 cases of arteriovenous fistulas in 1920 he found none which involved the vertebral vessels.

In discussing the problem of surgical treatment of traumatic lesions of the vertebral artery, Matas quoted the following remarks from Sanson's *Treatise on Traumatic Haemorrhages* which was published in 1836: "The vertebral artery cannot be ligated, on account of its great depth, nor compressed, because of the osseous canal which protects it; it can still less be cauterized. The wounds of this vessel are beyond the resources of art." As the years have passed and successful surgical attack upon these vessels has been achieved this feeling of pessimism has ceased. Nevertheless, the vertebral vessels still offer certain difficulties and hazards in operative approach because of their peculiar anatomical situation.

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The artery arises from the arch of the subclavian artery and passes at the lateral border of the longus colli muscle laterally and behind the common carotid artery posteriorly toward the vertebral bodies. The artery occasionally enters the transverse foramen of the second, third, fourth, or fifth cervical vertebra and even more rarely that of the seventh. Generally, however, it passes through the foramen of the sixth cervical vertebra and then traverses successively the other cervical vertebral foramina up to and including that of the atlas. It then passes beneath the superior oblique capitis and the posterior major rectus capitis muscles into the cranial cavity. The first, or extraforaminal, portion of the artery is about 3 centimeters in length. It is in close proximity to the thyrocervical trunk, carotid artery, jugular vein, vagus and phrenic nerves, and the anterior scalene and the longus colli muscles. The intraforaminal portion is difficult to approach because of the overlying bony covering of the transverse processes. The processes themselves are partly covered by the anterior, medial, and posterior scalene muscles and the longus colli and longus capitis muscles which take origin from these structures. More superficially the vessels are in part covered by the clavicle, the sternomastoid, sternothyroid and omohyoid muscles. The vein closely follows the artery throughout its course. The problem of approaching and controlling hemorrhage from these vessels has undoubtedly contributed to the difficulty of surgical cure of aneurysms and arteriovenous fistulas involving them.

During the past year I have successfully extirpated 4 extracranial vertebral arteriovenous fistulas. It is hoped that the technique which we have utilized and a discussion of the difficulties encountered in diagnosis and treatment may be of some help to others.

One of the cases was an arteriovenous fistula in the proximal free portion of the vessels, and

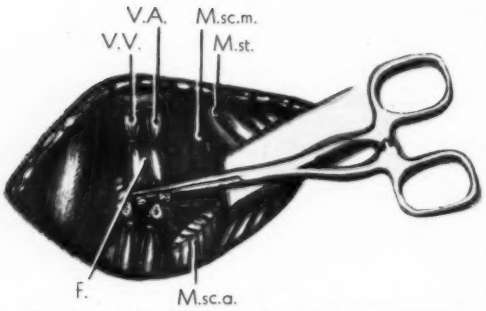

Fig. 1. Drawing showing the method of resection of arteriovenous fistula of intraforaminal portion of vertebral vessels. The transverse process of the sixth cervical has been removed, the artery and vein ligated above and below the fistula, and the fistula is ready for excision. *V.A.*, Vertebral artery; *V.V.*, vertebral vein; *F.*, fistula; *M. sc. m.*, scalenus medius; *M.st.*, sternomastoid; *M.sc.a.*, scalenus anterior.

3 existed in the intraforaminal part. This report is particularly concerned with the surgical treatment of lesions of the intraforaminal portion of the vessels. It has been found feasible to approach such lesions directly by resection of the overlying transverse process and to extirpate the fistula by excision, with ligation of the artery and vein proximally and distally, together with ligation of the spinal branches in the involved area (Fig. 1). The idea that a direct attack of this sort might be feasible sprang from a conversation with Colonel D. Elkin and Major M. H. Harris in February, 1945, concerning a patient in whom Major Harris had found it necessary to remove the overlying transverse process because of hemorrhage which he had encountered in attempting to dissect out and ligate the vessels between two of the transverse foramina.

CASE REPORT

CASE 1. A 25 year old soldier had been wounded on September 19, 1944, when he ran into the bayonet of a fellow American soldier during the confusion of enemy action. He sustained a penetrating wound of the right side of the neck just anterior to the mid portion of the sternocleidomastoid muscle. He attempted to control bleeding by digital pressure but lost a considerable amount of blood, although he did not lose consciousness or require transfusion subsequently. He was evacuated through several medical installations and was admitted on the following day to an evacuation hospital where the wound was débrided and closed with drainage. At this time it was ascertained that the wound extended down to the vertebral column. Initially he had hoarseness and dysphagia. These symptoms had cleared by the tenth day. At this time examination revealed evidence of an arteriovenous fistula in the neck. He was returned to the Zone of Interior and admitted to Mayo General Hospital on December 20, 1944. On admission his sole complaint was a buzzing which he felt and heard in his right ear.

Examination revealed a well healed transverse scar at the level of the thyroid cartilage. Here the carotid pulsation seemed definitely increased and a loud continuous bruit was present. On one occasion an examiner thought there was a continuous thrill in this area, but subsequently this was not found to be present. Deep pressure caused a diminution but not cessation of the bruit. The impression was held that the patient had a fistula between the carotid artery and jugular vein near their bifurcation. He was given intermittently prolonged compression of the carotid artery. Occasionally during such compression he had the subjective sensation that the buzzing in his ear ceased.

On January 12, 1945, exploration was carried out under intratracheal gas-oxygen-ether anesthesia. The old scar was excised and was extended anteriorly and posteriorly. The carotid artery, jugular vein, and vagus nerve were exposed. The jugular vein was slightly dilated and appeared to have within it eddies of arterial blood. No thrill was felt. The artery appeared normal. Both vessels were dissected upward and downward and the bifurcation and the first 3 branches of the external carotid vessels were visualized. Temporary occlusion of the common carotid, the internal and external carotid arteries, seemed to have little or no effect upon the bruit. The thyrocervical trunk and vertebral artery were isolated low in the neck. The bruit persisted during temporary occlusion of these vessels. The posterior cervical artery was also compressed without changing the bruit. The fistula could nowhere be identified and no thrill was palpable. I was at a loss to localize the lesion. Because of this and because of the presence of eddies of arterial blood in the jugular vein together with the fact that carotid compression preoperatively had some times caused diminution in the bruit and subjective cessation of the buzzing in the ear, I divided the carotid artery and jugular vein between ligatures. In retrospect this was an unwise procedure. Postoperatively the patient had the same buzzing, and not only was the bruit still present but there was also a slight but definite continuous thrill.

On February 17, 1945, the neck was again explored under gas-oxygen-ether anesthesia. At this time it was my feeling that the fistula undoubtedly existed in the vertebral vessels. The old scar was excised and the skin flaps were reflected. The sternomastoid was retracted laterally and the carotid artery, jugular vein, and vagus nerve were identified. Dissection was carried down to the sixth cervical vertebra and here a definite thrill could be felt. Because of the scarring from the previous operation, it was nece-

sary to make a longitudinal skin incision downward in order to secure adequate exposure, and also to transect the sternocleidomastoid muscle. The vertebral artery and vein were isolated below the sixth transverse foramen and occlusion of these vessels diminished but did not obliterate the thrill and bruit. This phenomenon, it was felt, was due to the extensive collateral circulation present. The transverse process of the sixth cervical was rongeured away so as to open the foramen. Pressure upon the vessels in this area caused complete cessation of thrill and bruit. The artery and vein were divided between ligatures proximal to the sixth foramen and by gently lifting them away from the foramen it was possible to demonstrate a communication between the vessels in the foramen itself. Three large spinal branches were encountered which were isolated and divided between ligatures. One large branch which seemed to perforate the vertebra was torn across and this was followed by brisk bleeding which was controlled by insertion of a piece of muscle. The vessels were traced up to the foramen of the fifth cervical vertebra where they were individually ligated. The specimen was then excised. There was now no longer any bruit or thrill. There was no bleeding. The wound was repaired in layers with fine black silk sutures. The specimen was torn somewhat in its removal and the pathologist, though unable to demonstrate a definite fistula, found that at one level more than a third of the wall of the artery was necrotic, distorted, and shredded.

The patient made an uneventful recovery. He had no return of bruit or thrill and was discharged from the hospital 2 months after operation.

CASE 2. The patient was a 22 year old soldier who was admitted to Mayo General Hospital on October 2, 1944, because of an arteriovenous aneurysm in his left arm. He had been wounded on August 17, 1944. He had been found to have evidence of an arteriovenous aneurysm and on September 9 subcutaneous rupture had occurred with enormous swelling of the arm and shoulder. Paralysis of the left upper extremity, which had up to this time been incomplete, was now complete and the radial pulse disappeared. He had extreme throbbing pain in the entire extremity and had required narcotics for sedation. On examination he was found to be in great pain. There was marked swelling of the left shoulder and arm. There was no thrill but a loud continuous bruit was present just beyond the axillary fold. This was even louder just proximal to the swelling, and it was noted that there was a continuous thrill and bruit also present in the left side of the neck. His left brachial, radial, and ulnar pulses were absent. There was anesthesia of the entire left forearm and absence of motor power in the entire left upper extremity. His blood pressure was 130/60 and pulse was 90. The impression was held that the patient had an axillary arteriovenous aneurysm with a large false sac which had undergone subcutaneous rupture. It was apparent that at least part of his paralysis was due either to pressure or to ischemia.

It was recognized at the time that the presence of the thrill and bruit in the left side of his neck very likely indicated the presence of a second arteriovenous fistula. There was a minute scar in this area and the patient stated that approximately 10 months prior to his recent injury he had received a minor shell fragment wound in the neck which had caused no bleeding or discomfort and for which he had not sought medical aid. Because of the impairment of his circulation and especially because of the severe pain in his left hand, a sympathetic block was done. This procedure resulted in some improvement in circulation and in considerable relief of pain. For this reason a dorsal sympathectomy was done on October 11. On October 15 there was a rupture through the macerated skin of the axilla. Fortunately, this was immediately plugged by a portion of the thrombus. The following day the aneurysm was explored. There was a very large sac which was poorly organized and largely filled with thrombus. The vein had been thrombosed. It is of interest that the continuous bruit in this area had changed to a systolic one 6 days previously. There was a large rent in the brachial artery, the involved portion of which was excised. The patient had been febrile on admission and had had local heat and redness suggesting infection. At the time of exploration, frank pus was found surrounding the aneurysm. The patient made an uneventful convalescence.

The thrill and bruit persisted in the left side of the neck. It was impossible to obliterate them by direct pressure and the exact location of the fistula could not be determined. On January 18 a low oblique incision was made in a skin crease of the left side of the neck. The carotid and jugular vessels were identified, and no fistula was found between them. A thrill could be felt posterior to these vessels. The anterior and a portion of the medial scalene muscles were divided. The vertebral artery and vein were identified in their first portion and here were present a thrill and bruit which ceased with temporary occlusion of these vessels. They were divided between ligatures. There was no longer a thrill or bruit, and although it was realized that a cure was by no means certain, it was decided to close the wound rather than to attempt to extirpate the fistula in the intraforaminal portion. The operation had consumed nearly 2 hours. After operation the thrill and bruit recurred, and exploration was again carried out on February 28. The anterior third of the old wound was excised and the incision was extended obliquely upward. The sternomastoid was retracted. The carotid artery, the jugular vein, and the vagus nerve were identified. In the region of the sixth vertebra a thrill was felt. The muscles arising from this transverse process were detached. The vertebral artery and vein were isolated just proximal to the sixth foramen and here a bulge was noted as the vein entered the foramen. The vessel in this area was nearly 1 centimeter in diameter. The transverse process was carefully removed and the vessels were dissected out. A spinal branch was divided between silver

clips. The vessels were dissected up to the fifth foramen and because of difficulty in separating the artery and vein here, they were twisted and ligated *en masse*. There was no bleeding. The bruit and thrill were gone. Examination of the excised specimen showed the presence of an arteriovenous fistula. The wound was closed in layers with fine silk sutures. The patient made an uneventful recovery and has had no recurrence of his bruit or thrill. The patient has had some return of nerve function in his left upper extremity, but unfortunately proper neurosurgical treatment has been delayed by minimal recurrent infection in a portion of the axillary wound.

CASE 3. A 26 year old soldier had been wounded by a shell fragment on February 16, 1945. He lost consciousness and bled freely from a wound in the left side of the neck. He developed a large soft hematoma and had a transient partial paralysis of the left arm. X-ray examination revealed a large foreign body about 2 by 2 centimeters in size anterior and lateral to the lower cervical vertebrae. On February 16 the foreign body was removed. A fracture was noted in the transverse process of the sixth cervical vertebra. He was returned to the Zone of Interior and was admitted to Mayo General Hospital on May 3. Here for the first time it was noted that he had evidence of an arteriovenous fistula in the left side of his neck.

The patient stated that he had regained most of the function in his left arm. He said that he became somewhat hoarse whenever the weather was damp. A scar was present on the left side of the neck along the anterior border of the sternocleidomastoid. Here there was an abnormal pulsation which was palpable but not visible. There was a continuous thrill and a loud harsh continuous murmur. This phenomenon was transmitted over the entire left side of the neck and over the whole precordial area. It was loudest immediately above the clavicle. Deep pressure at this point caused cessation of thrill and bruit. With this procedure the pulse slowed and the blood pressure rose from 120/62 to 134/82. He had evidence of returning function following a partial paralysis of his brachial plexus. It was felt that the patient had an arteriovenous fistula between the carotid artery and jugular vein.

On June 14, exploration was carried out under intratracheal gas oxygen ether anesthesia. The old wound was excised. The sternocleidomastoid was retracted posteriorly. The carotid artery, jugular vein, and vagus nerve were identified. Occlusion of these vessels did not obliterate the thrill or bruit. One could palpate posteriorly to the vessels a large vertebral artery and vein and could demonstrate that compression of these vessels against the vertebral body just proximal to the sixth transverse process caused cessation of bruit and thrill. They were exposed. The vertebral vein was about 5 millimeters in diameter and just as it entered the sixth foramen was nearly 1 centimeter in diameter. The transverse process was carefully removed. The vertebral artery and vein were divided between ligatures proximally and then they were carefully dissected free up to the level of the fifth foramen. Here they were divided between transfixing ligatures. A large spinal venous branch had been successfully divided between clamps and was ligated. There was some bleeding from an arterial spinal branch within the foramen which was controlled by packing with fibrin foam. Examination of the excised specimen showed a fistula to exist between the vertebral artery and vein about 4 millimeters in diameter. This fistula occurred in that portion of the vessels which lay within the sixth transverse foramen. The patient made an uneventful convalescence. His neurological complaints in the upper extremity cleared and he was discharged from the hospital 6 weeks after operation.

CASE 4. A 26 year old officer was admitted to the Mayo General Hospital on September 11, 1945, with a diagnosis of carotid-jugular arteriovenous fistula. He had been wounded by shell fragments on November 5, 1944. Both eyes had been injured with resultant enucleation of the right eye and reduction of vision in the left to 3/400, correctible to 20/400. He had sustained a hemothorax and perforation of the diaphragm and duodenum. A laparotomy had been performed with repair of the diaphragmatic and duodenal lacerations. The hemothorax had cleared following several thoracenteses.

About a month after injury a thrill and bruit had been noted in the right side of his neck. The patient had been aware of a continuous roar in his right ear from the time of injury but at first had thought that this was simply the result of the concussion. He had noted no other symptoms and had no precordial distress, edema, cyanosis, or dyspnea. He stated that in learning to walk with little or no vision the buzzing in his right ear had been a handicap in orientation.

Examination revealed a healed wound in the right midscapular area and another in the right sternoclavicular area. Just above the sternoclavicular region a prominent continuous thrill was palpable and in this area a loud continuous bruit was heard. Direct digital compression here caused a cessation of bruit and thrill and of the carotid pulsation. During such compression the brachial pulse was unimpaired. With compression, the pulse slowed from 60 to 43 per minute, and the blood pressure changed from 130/70 to 132/88. It was felt that the patient had a carotid-internal jugular arteriovenous fistula. The patient was given repeated digital occlusion of the carotid artery until he could stand such compression for 30 minutes without symptoms. On September 24, exploration was carried out under intratracheal gas-oxygen-ether anesthesia. The old scar was excised and the skin flaps were reflected. The sternocleidomastoid was retracted laterally, and the carotid sheath was opened. The internal jugular vein was about 1.5 centimeters in diameter, and a continuous thrill was palpable. This thrill disappeared with strong digital pressure over the vein, about 1.5 centimeters superior to the clavicle. The

anterior and mesial surface of the vein was gently dissected free. The vagus nerve and the carotid artery which lay mesial to the vein were isolated and were found to be normal. It was apparent then that the fistula involved the underlying vertebral artery which could be felt to be pulsating strongly. Since the fistula was well above the clavicle it was not felt necessary to resect the clavicle in order to expose the subclavian artery. The sternocleidomastoid muscle was divided in order to facilitate exposure. Since the jugular vein was firmly adherent to the underlying structures, it was necessary to mobilize it in order to expose the vertebral vessels beneath. A tape was placed about the vein distal to the fistula. Isolation of the vein proximal to the fistula was difficult because of scarring and just as it was about completely freed a small tear was made into the fistula itself. Bleeding was easily controlled by digital compression. The vein was divided between ligatures proximally and distally. A clamp was placed across the fistula so as to occlude the opening. It was then noted that a thrill and continuous bruit were still present, and it was obvious that the vertebral artery opened not only into the overlying jugular vein but also into the vertebral vein which lay posterior to it. The vertebral artery and vein were ligated proximally and distally, and the specimen was excised. Exposure was quite adequate throughout. The muscle, fascia, and skin were approximated with interrupted silk sutures. In Figure 2 the fistula, as it was seen at operation, is illustrated semidiagrammatically.

The patient did well postoperatively. The buzzing in his ear was gone and he walked with more ease and assurance. He was transferred to another hospital for further instruction necessitated by his blindness.

It will be noted that in all 4 of the cases reported the preoperative diagnosis was erroneous. In all of them the impression was held that the fistula was between the carotid artery and the jugular vein. In Cases 3 and 4 it is difficult to see how any other diagnosis could have been made since the bruit and thrill could be readily obliterated by digital compression which occluded the carotid pulsation. In Cases 1 and 2 it is felt in retrospect that strong suspicion should have been held that the fistula involved the vertebral vessels because of the inability to obliterate the bruit by digital compression. The diagnosis of vertebral arteriovenous fistula will necessarily always be difficult. It should always be borne in mind whenever digital pressure does not bring about cessation of bruit and thrill. Furthermore, it may be anticipated that in certain instances where the clinical evidence suggests a lesion of the carotid jugular vessels,

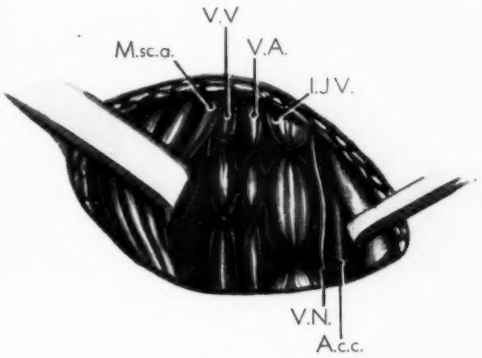

Fig. 2. Arteriovenous fistula between first portion of the vertebral artery and the vertebral and internal jugular veins. *M.sc.a.*, Scalenus anterior; *V.V.*, vertebral vein; *V.A.*, vertebral artery; *I.J.V.*, internal jugular vein; *V.N.*, vagus nerve; *A.c.c.*, common carotid artery.

the fistula will be found in the vertebral vessels.

At the time of operation confirmation of the diagnosis of vertebral arteriovenous fistula can generally be made by a process of exclusion. When the other vessels in the neck are isolated and temporarily occluded without causing cessation of bruit and thrill one must be strongly suspicious that the lesion involves the vertebral vessels. It should be remembered, however, that occasionally the collateral circulation is so ample that the bruit will persist even during proximal occlusion of the vertebral artery and vein. This is exemplified by Case 1. The error made in the treatment of Case 1 should not be repeated. There is little chance of affecting a cure by ligation of vessels in the neck unless the fistula itself is identified and is excised. The one exception is a fistula between the internal carotid artery and vein. These vessels ordinarily have no branches and hence fistulas between them can be cured with considerable regularity by proximal ligation, a procedure which in almost all other instances is futile.

With regard to the surgical approach to the vessels it should be emphasized that adequate exposure is necessary. In all 4 of my patients there was present at the time of operation some pre-existing scar which was excised or modified. From my experience I feel that an oblique incision in one of the skin creases is

probably ample for exposure of the first and second portions of the vertebral vessels. I have had no experience with fistulas in the distal intraforaminal portion nor between the atlas and the cranial cavity. The sternomastoid should be either retracted or divided. The carotid artery, jugular vein, and vagus nerve are retracted mesially. The anterior scalene muscle should be divided. Should a lesion exist close to the origin of the vessels from the subclavian artery, it would be wise to resect the inner third of the clavicle in order to have control of the subclavian vessels. If a lesion is present in the intraforaminal portion, direct exposure and excision is feasible by careful removal of the overlying transverse processes. Before this step is taken the slips of the scalene muscle and the longus colli and longus capitis muscles, which are attached to the transverse processes, must be stripped away by sharp dissection and the use of a periosteal elevator. In the 3 cases described it was possible to remove the overlying transverse processes without injury to the underlying vessels by gently depressing the vessels while the process was excised with a small rongeur. It may be anticipated that some difficulty will be encountered in the handling of the spinal branches. In my experience these frequently can be isolated and either ligated with silk or clipped with a silver clip. In case this was not possible bleeding was controlled either by packing with muscle or with fibrin foam. Because of the rich supply of collateral vessels in this area, it is felt that a permanent cure can rarely be anticipated by any other surgical means than excision with ligation of the involved vessels.

In none of the cases reported was there any evidence of cerebral circulatory difficulty. All of the patients had been given prolonged carotid compression prior to operation until they could withstand such compression for approximately a half hour without symptoms. In several cases, electroencephalographic studies were carried out prior to operation, both at rest and during compression of the artery. It will also be noted that one of the patients reported had a division of the common carotid in addition to ligation of the vertebral artery. The abundant collateral circulation about the vertebral vessels in the neck and the communication in the circle of Willis would lead one to judge that cerebral ischemia would rarely follow ligation. It is important to record, however, that I have met with disaster in one case in which aneurysmorrhaphy of a large subclavian aneurysm involved ligation of no vessel supplying the brain except for one vertebral artery. The patient died the following day and postmortem examination revealed homolateral cerebellar necrosis without evidence of thrombosis or embolism.

CONCLUSION

1. Vertebral arteriovenous fistulas are relatively rare, and the proper localization of such lesions is difficult.

2. Four cases which illustrate the difficulty in diagnosis and some of the difficulties which may be encountered at the time of operation are presented.

3. A method of dealing with intraforaminal lesions of the vertebral vessels by their direct exposure and excision after resection of the overlying transverse processes is described.

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TENDON TRANSPLANTATION IN THE HAND

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TENDON transplantation is the free grafting of a tendon for the purpose of filling a tendon defect or replacing a destroyed tendon. A tendon defect is either due to destruction of a tendon from trauma or disease, or due to contraction of the muscle belly after severance of its tendon. In either case, return of function can be expected only if the defect is bridged with a suitable tissue graft. If the defect is due to infection, one ought to delay the operation at least 6 months. However, since the advent of penicillin, the waiting period can be shortened 3 months. Penicillin should be administered 2 days preoperatively and continued for at least 4 days postoperatively.

For the bridging of tendon defects, various autogenous or hemogenous tissues, such as fascia, cutis, tendon, and even heterogenous material, such as paraffined silk have been recommended. Among the pioneers of this work are Kirschner, Bier, Lexer, E. Rehn, Lange, Bunnell, Koch, Mason, Mayer, and Iselin. According to the present consensus, autogenous tendon tissue is favored as graft material. The graft sources are the palmaris longus, the long extensor tendon of the 4th and 5th toes, and—under certain circumstances to be mentioned later on—the flexor sublimis tendon.

In every tendon grafting, aside from the regeneration of the tendon tissue, the rôle of the gliding tissue is of interest and importance. This tissue, called paratenon, is a loose, fatty meshwork rich in elastic fibers. The paratenon not only provides the gliding mechanism but the bulk of the nutrient vessels and nerves of the tendon.

The tendon sheath is a closed sac containing fluid. It is found whenever the tendon changes its direction and serves as a fluid buffer to diminish friction at this point. The sheath consists of a parietal and visceral layer. Both layers are connected with each other by the mesotenon which carries the nutrient vessels and nerves. Thus, it becomes evident that a tendon graft has little chance to survive and to function unless the gliding tissue of the host area is preserved, or, if absent, the gliding tissue is also transplanted. Hence, if the paratenon or the tendon sheath of the graft bed is absent, a paratenon covered tendon graft must be transplanted. So, for instance, in using one of the long extensor tendons of the toes as a graft, great care should be taken to preserve the loose areolar tissue which surrounds the tendon. It not only should be preserved but firmly held in place with a temporary suture at the extreme end of the graft. The paratenon, like the periosteum of a bone graft, gains quick access to the circulation of the surrounding tissue and is now able to re-establish the interrupted blood supply within the tendon graft. If the paratenon of the graft cannot be preserved, the latter must be taken from other sources as a free graft, and be wrapped around the tendon graft and fastened with a temporary silk suture. The paratendinous tissue differs from ordinary fat tissue. It is a loose areolar fat tissue which is found over the triceps tendon or in the interspace between the fascia lata and the muscle fascia.

Opinions still differ as to the way of regeneration of a tendon graft. One group of investigators believes that the graft is substituted by fibrous tissue which is derived from the surrounding tissue. Another group believes in a true or at least a nearly true regeneration of the tendon graft coming from the paratenon or due to metaplasia. All agree that the functional stimulus promotes quicker and better regeneration. Whatever the way of regeneration may be, it has been sufficiently proved, experimentally as well as clinically, that a grafted tendon segment under preoperative and postoperative care, can regain function to transmit the contractile force of its muscle. However, it also should be pointed out here that stiff joints which cannot be

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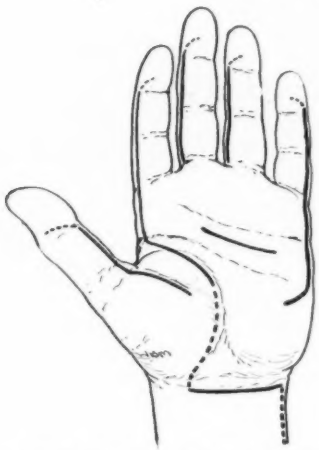


Fig. 1. Correct incisions in the hand. At the fingers the lateral incisions are the best; in the palm incisions should be parallel with the creases; in the wrist incisions should be transverse. Dotted lines indicate additional incisions and combinations.

Fig. 2. Bayonet incisions of the hand after Mason.

moved passively are a strict contraindication to any tendon repair unless mobilization is possible either by physiotherapy or by operative manipulation.

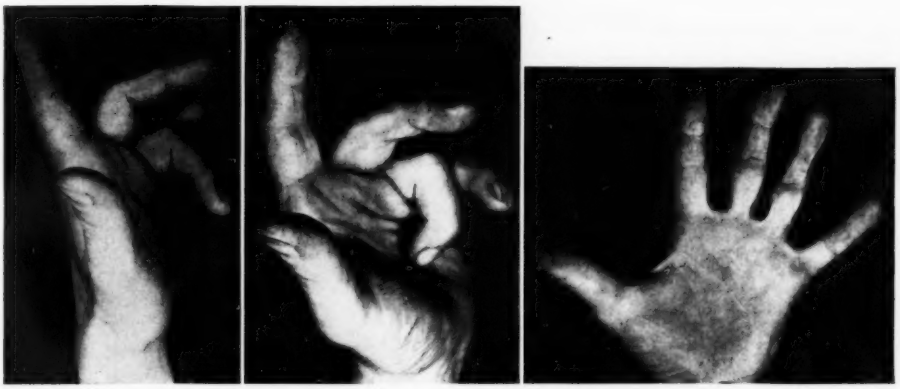
EXPOSURE OF THE GRAFT BED

The operation is performed under general anesthesia. A blood pressure cuff is applied and inflated to obtain a bloodless field. If the dissection lasts longer than 1 hour, the pressure should be released temporarily. For locating the destroyed or divided tendons, incisions are made which should be placed in such locations as not to interfere with the function of the hand (Figs. 1 and 2). At the fingers the lateral or bayonet incisions are the best. In the palm the incision should be parallel with the flexion crease. In the wrist the incision should be transverse. If for better exposure additional incisions are necessary, they should be placed according to the aforementioned principles. So, for instance, the incisions of the fingers and palm can be combined in suitable places. At the wrist a transverse incision can be enlarged by secondary longitudinal incisions on either side.

In locating the divided or destroyed tendons, all surrounding adhesions should be divided. The surrounding scar tissue must be thoroughly excised until healthy tissue is left. Care should be taken not to injure the nerves. The handling of tendons and nerves should be as gentle as possible. They should be covered with moist gauze; if grasped with instruments, only their divided ends and the parts to be sacrificed are clamped with a mosquito forceps. All tendon tissue which is devitalized, cicatricial, or frayed is excised with a very sharp knife until normal looking tendon tissue is encountered, no matter how large the resulting defect. The blood pressure cuff is now deflated and bleeding checked by thorough hemostasis.

SOURCE AND FIXATION OF THE GRAFT

If the tendon defect is in the palm and involves the sublimis and profundus tendon it is generally agreed to bridge only the gap in the profundus tendon, to avoid having grafts and suture lines at the same level in close approximation favoring adhesions, and thus prevent the profundus tendon from acting on the terminal phalanx. The source of the graft is the proximal segment of the sublimis tendon; while its distal segment is excised. The graft, after its fixation to the tendon stumps, should be under moderate tension. To follow Koch's advice, the hand should be flexed in the wrist to an angle of 120 to 110 degrees. and the fingers semiflexed



a b c

Fig. 3. a, Patient, aged 37 years, had a subcutaneous rupture of the flexor profundus tendon of the 4th finger of the right hand. A heavy weight had fallen on the extended hand and had bent the 4th finger extremely backward. Upon operation, 2 months after the accident, the profundus tendon was found severed near the insertion of the distal phalanx. The proximal stump had retracted into the palm. It was impossible to pull the tendon to its insertion place, hence, a tendon graft was used (the long extensor tendon of the 4th toe). b and c, Patient regained almost all motility of the injured finger within 5 months.

at the metacarpophalangeal joints to an angle of 120 degrees. The width of the resulting gap of the profundus tendon is now measured, and this distance marked off on the proximal segment of the flexor sublimis. The latter tendon is severed at that point and excised. The excised part is laid between the ends of the profundus tendon and fastened to them with a tendon suture. If a sublimis tendon is not available for grafting, the tendon of the palmaris longus or the long extensor tendon of the 4th or 5th toe may be utilized.

a b c

Fig. 4. a, Boy, aged 13 years, had a laceration of the flexor tendons of the 4th finger in level of the first interphalangeal joint. Sublimis and profundus tendons were sutured primarily. Operation was performed elsewhere. After 3 months it became evident that the profundus tendon was not functioning. An exploratory operation was advised and carried out. The profundus tendon was found severed. The proximal stump had retracted into the palm. The distal segment together with the repaired sublimis tendon formed a mass of scar tissue. No evidence of a tendon sheath was found. The distal end of the profundus tendon was radically excised. The excision included the remnants of the annular ligament (pulley of the middle phalanx). The sublimis tendon was also removed and used as a graft to repair the defect in the profundus tendon and to replace the annular ligament. b and c, Patient regained almost full motility in 7 months.

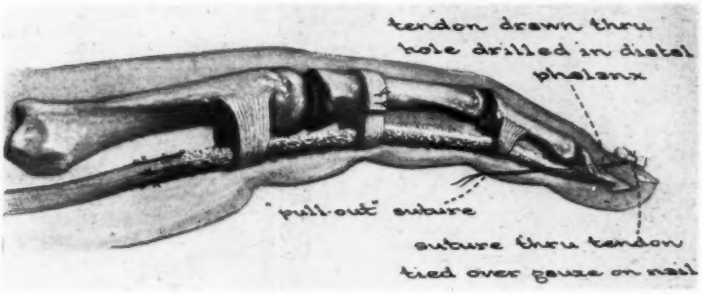


Fig. 5. Grafting of a flexor tendon and reconstruction of an annular ligament after Bunnell. Loose areolar fat tissue is wrapped around the tendon graft to provide the gliding tissue. The distal end of the graft is fastened directly to the bone. A hole is made through the terminal phalanx large enough to allow the tendon graft to pass snugly through it. The canal is drilled obliquely from the proximal part of the volar surface of the bone distal to, but not through, the nail. The hold suture of the graft is made removable from the outside. A fine stainless steel wire needle on each end is followed in mattress suture fashion through the end of the graft. In order to remove the wire 3 weeks later, a second wire is threaded through the proximal loop of the suture wire and then its two ends are threaded together on a straight needle and led proximalward out through the skin and left there. This is a pull-out wire. The two needles of the suture wire are passed through the bone canal and through the nail, thus pulling the distal part of the graft into the bony canal. The two ends of suture wire are tied over a small roll of gauze or a rubber button. The annular ligament is reconstructed with a piece of tendon graft that is passed around the phalanx with a sharply curved aneurysm needle; the ends of the new annular ligament are overlapped and sutured together. The suture would come to lie at the side of the bone and not over the tendon. The proximal end of the tendon graft is sutured to the proximal end of the profundus tendon.

The volar side of the finger is the region where tendon grafts are used frequently (Figs. 3 and 4). It is generally agreed (Iselin, Koch, Mason, Cutler, and others) that a primary repair of tendons, severed within the tendon sheath, should be attempted only under the most favorable circumstances. The slightest infection or irritation following the repair will cause scar formation within the sheath and adherence of the tendons to the sheath, so that not only the immediate result will be spoiled but also secondary repair becomes more difficult. Bunnell went even farther by recommending secondary repair and substitution of the entire divided tendon by a graft to avoid a suture line within the

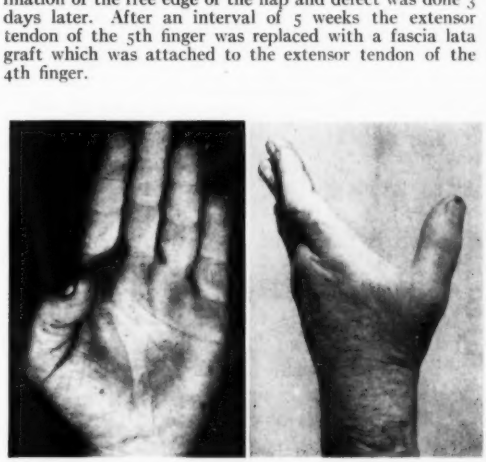
Fig. 6. Method of attaching a tendon graft to the distal phalanx after Koch. From a volar extension of the original incision a small hole is drilled transversely through the phalanx. From a small incision on the other side of the finger a Reverdin needle or suture carrier armed with the tendon graft is inserted through the bone carrier and withdrawn. The instrument is reinserted from the original incision, passed in front of the phalanx to escape through the second incision. It again is armed with the tendon graft and withdrawn. The graft is then united on the volar surface of the phalanx by an end-to-side union.

a b c d

Fig. 7. a, Patient's arm was caught in a combing machine. Extensive loss of surface tissue on dorsal side of the hand. Exposure of the tendons and loss of extensor tendon of 5th finger. Note exposure of the 5th metacarpal bone. b, Abdominal flap was raised from the opposite side of the abdomen at the time of the wound excision. It was returned to its original site for developing adequate circulation. Two weeks after the operation the flap was transferred to the right hand. The pedicle of the flap was partially severed 10 days after transfer of the flap. It was entirely severed on the 14th day. Adjustment and approximation of the free edge of the flap and defect was done 3 days later. After an interval of 5 weeks the extensor tendon of the 5th finger was replaced with a fascia lata graft which was attached to the extensor tendon of the 4th finger.

sheath. Hence, secondary repair is indicated in the majority of cases. In a great number of cases the muscle belly contracts in the meantime to such a degree that the tendon stumps cannot be united with each other, and the gap between them must be bridged with a tendon graft.

If the sublimis and profundus tendon are divided, it is advisable to bridge only the profundus tendon for the reasons mentioned. Graft sources are the proximal segment of the divided sublimis tendon or, if the latter is not available (at the thumb for instance), the palmaris longus or the long extensor of the 4th or 5th toe. From a lateral or bayonet incision the adherent tendon stumps are mobilized. The distal segments are found after opening the tendon sheath close to its bone attachment. In the level of the proximal phalanx three tendons are found: the profundus tendon and two slips of the sublimis tendon. The sublimis tendon and its two slips are excised. The next step consists in exposing the proximal tendon stumps, which usually have retracted into the palm or wrist. They are located from a separate incision in the palm or over the wrist. The



a b

Fig. 8. a, Patient, aged 49 years, fell and bumped radial side of left hand on a cable. The hand was swollen. There was no impairment of function of tendons of thumb. About 1 month later patient felt a sharp pain in region of tabatière after extending thumb. The thumb snapped into flexion and could not be extended. Diagnosis was rupture of extensor pollicis longus. Upon operation extensor pollicis longus was found severed about 2 inches proximal to metacarpophalangeal joint. Distal tendon stump was freed. Tendon sheath was obliterated with scar tissue. Proximal tendon had retracted into distal third of forearm. There was a gap of about 4 inches between the two tendon segments. Proximal tendon segments could not be pulled downward. A tendon graft taken from long extensor tendon of 4th toe was used to bridge defect. Immobilization of thumb in extension abduction for 3 weeks. b, Thumb regained almost full motility within 3 months.

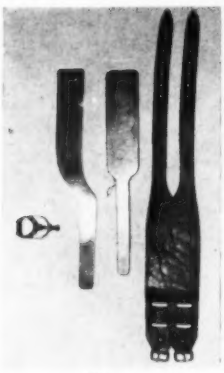


Fig. 9.

a

Fig. 10. b

Fig. 9. A finger splint which permits independent active motion exercises of the interphalangeal joints of the finger.

Fig. 10. a, The splint depicted in Figure 9 in active use. The flexor profundus tendon of the 4th finger was replaced by a tendon graft. When active exercises were instituted without the splint, the patient moved, mainly, the joint which was movable before the operation, namely the metacarpophalangeal joint. To force the other joints to active exercise the splint is applied, a clamp is slipped over the finger, and the splint holds the metacarpophalangeal joint immobilized. The patient now moves the first interphalangeal joint. b, Clamp immobilizing middle phalanx; patient can now exercise the terminal joint.

tendons are freed from scar tissue and pulled into the wound. If the sublimis tendon is to be used as a graft and its proximal stump has been located in the palm, a third incision is made over the wrist. From this incision the sublimis tendon is located and pulled out of the wound and severed. If the severed tendon is found to be without its gliding tissue, the latter must be provided and wrapped around it as described. If the sublimis tendon is not available, the tendon of the palmaris longus or one of the long extensor tendons of the 4th or 5th toe are utilized. The graft is now sutured to the distal stump of the profundus tendon. In the majority of cases, due to excision or destruction of the tendon sheath, a new annular ligament must be reconstructed to hold the tendon in place over the volar surface of the fingers. This is done according to Bunnell's plan as follows (Fig. 5): Another tendon graft available from the sources mentioned above is split and one of the slips is passed around the phalanx at the site of the maximum tension with a sharply curved aneurysm needle. The tip of the aneurysm needle is kept in close contact with the bone so as not to include any of the digital vessels or nerves, or the extensor tendon. The ends of the new annular ligaments are overlapped and sutured together. The suture should come to lie at the site of the bone and not over the tendon.

If the distal tendon segment is too short to permit a secure suture, it should be sacrificed and the graft fastened directly to the bone. This can be done according to Koch (Fig. 6) or Bunnell's method (Fig. 5). I have tried both methods but consider Koch's method as the more secure attachment. Then follows closure of the finger wound in layers. Koch points out that it is a real advantage to be able to close the incision in the finger while the finger is still extended, and before the suture of the graft to the profundus tendon in the palm is carried out. To close an incision extending along the greater part of the finger while the finger is supported in flexion and while one is attempting to avoid tension on a newly sutured tendon, is a difficult task. After the finger wound is closed, the proximal end of the graft is attached to the proximal segment of the tendon under moderate tension. If the proximal tendon stump was ex-

posed from a separate incision, the intervening skin, subcutaneous tissue bridge is tunneled. A probe is led peripherally into the finger wound; the free end of the graft is armed with a mattress suture, which is threaded through the eye of the probe. The latter is now withdrawn pulling the tendon graft through the tunnel into the proximal wound where it is sutured to the proximal tendon stump. The remaining wound or wounds are closed in layers and the hand carefully supported until the immobilizing splint is applied.

On the dorsum of the hand, grafts are less often indicated, since there is less tendency of contraction of the muscle bellies, and even if it does occur the juncturae tendinum connecting the tendons with each other are quite often strong enough to transmit the function of an uninjured tendon to the neighboring injured tendon. If, however, a gap must be bridged, it is best bridged with a long extensor tendon of the 4th or 5th toe (Figs. 7 and 8). If the gap happens to be in the extensor tendon of the third finger, Mayer proposes to transfer one of the two extensors of the index finger to the third finger through a subcutaneous tunnel and to fasten it there to the distal tendon segment. If the gap happens to be over the middle phalanx of the finger, the defect ordinarily does not need to be bridged. The extensor tendon divides here into three slips. It rarely happens that all three slips are missing, and it is my experience that the two lateral slips or even one of them is sufficient to carry on most of the function. If, however, the gap happens to be in the dorsal aponeurosis of the proximal phalanx or the proximal interphalangeal joint, the gap must be bridged. The graft to be used in this region is a piece of fascia lata with a thin covering layer of areolar fat tissue attached. The graft is folded so that the two "raw" layers are upon each other. The outside of the graft all around consists now of covering areolar fat tissue.

AFTER TREATMENT

This is as important as the operation itself. The healing process of a tendon wound passes through two stages: the proliferative and the formative stage. The first stage lasts about 2 weeks. During this time the tendon stumps become united by a connective tissue callus. The latter is then gradually converted into tendon. Mason and Allen, who did considerable experimental work on this subject, came to the conclusion that function during the first phase, did not accelerate the development of the tendon callus. On the contrary, function appeared to be harmful insofar as it caused more reaction of the surrounding tissue and weakening of the union. Later on, however, after the first callus has formed, restricted use of the tendon caused but slight irritation with rapid increase in tensile strength of the union.

It is common belief among surgeons, that motion, if started early, will prevent the adhesion of tendons. However, according to above findings and clinical experience, the opposite seems to be the case. Early motion, restricted or unrestricted, favors adhesions. The latter are due not only to irritation of the surrounding tissue, but also to disturbance of the vascularization of the tendon. This is a factor which should be pointed out strongly. The bulk of the blood supply of a tendon outside its sheath is derived from the paratenon. It is only natural that a tendon operation causes some disturbance of the local circulation, since it is almost inevitable to leave the intimate connections of the paratenal and surrounding tissue undisturbed. This is particularly true in tendon grafting. If, however, the involved tendon is adequately immobilized, the interrupted blood supply of the paratenon and tendon will become reestablished. Early motion, however, may disturb vascularization leading to necrosis of the gliding mechanism and formation of adhesions.

I prefer the molded plaster cast splint to any other kind of splint. The hand is placed in such a position that the suture line is under a minimum of tension.

After 2½ weeks, splint and dressings are taken off temporarily, and the sutures removed. Forearm and hand are placed, from now on, daily in a warm saline bath and active motion exercises encouraged. The splint is reapplied after the bath. Three weeks after the operation the splint is discarded and

local heat (baking, warm water bath, whirlpool bath), massage, and passive exercises are added to the active exercises. In addition to physical therapy, occupational therapy is of great importance. One of my patients developed a simple but very effective splint for exercising those joints which move freely (Figs. 9 and 10). Since introduction of this splint some of my patients regained full motility of the fingers without the aid of physical therapy.

SUMMARY

The use of tendon tissue is recommended for tendon grafting. The regeneration of the tendon graft and its gliding tissue is discussed. Various methods and examples of tendon grafting in various regions of the hand are described, together with the after treatment.

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FURTHER EXPERIENCES WITH PANLARYNGECTOMY FOR ADVANCED CARCINOMA OF THE LARYNX

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IN 1943 panlaryngectomy was described as a procedure indicated in advanced carcinoma of the larynx (or immediately adjacent portions of the hypopharynx) where metastases had not yet extended beyond the cervical lymph nodes (1). The operation had been reported in detail (Fig. 1) and in principle consists of resection *en masse* of the larynx, base of tongue, upper 3 to 4 centimeters of trachea below the larynx, and upper portions of each thyroid lobe and anterior portion of most of the cervical esophagus. The muscles inserting on the thyroid cartilage, as well as those of the infrahyoid group, are resected with the above; they are transected as far away from their thyroid cartilage and hyoid attachment as possible. The chain of cervical lymph nodes on each side are dissected from below upward in such a manner that they are freed from all connections except those attaching them to the thyroid cartilage and adhering muscles. In this fashion they are removed *en masse* with

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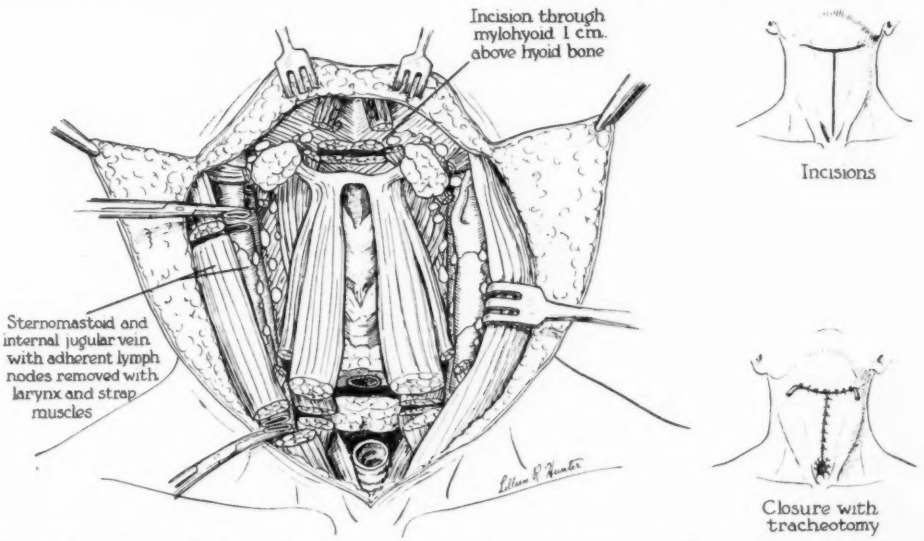
Incision through mylohyoid 1 cm. above hyoid bone
Sternomastoid and internal jugular vein with adherent lymph nodes removed with larynx and strap muscles
Incisions
Closure with tracheotomy

Fig. 1. Schematic illustration of panlaryngectomy. The strap muscles are transected low in the neck; both bellies of the digastrics are cut; the submaxillary glands and nodes are dissected free *en masse* and reflected downward and toward the midline; the mylohyoid muscle is cut through into the pharynx. When a jugular vein and adherent carcinomatous nodes are to be excised with the larynx and attached muscles, the vein is transected low in the neck and as high in the neck as possible. To obtain a block excision, the sternomastoid is also freed by transection near its insertion and near its origin, and removed *en masse* with the other structures excised. The trachea is divided below the isthmus of the thyroid and the lower end is brought up to level of skin. The upper half of the thyroid, or the upper portions of both poles, adherent to the trachea is removed. At the completion of the operation the trachea is sutured to the skin with interrupted silk sutures; small rubber drains are inserted into each extremity of the horizontal portion of the incision, and stab wounds may also be made in the lower portions of the neck on each side for drainage. Insets show T incision and method of closure. (*Surg. Gyn. Obst.*, 1943, 76: 391.)

TABLE I.—SUMMARY OF 7 PATIENTS WHO RECEIVED PANLARYNGECTOMY FOR ADVANCED LARYNGEAL CARCINOMA

Case	Patient Age	History	Date of panlaryngectomy and findings	Postoperative course
VI.	C.L. (329102) 54 years.	Dysphagia and hoarseness for 2 years. April 1943, 36 x-ray treatments to larynx. Moderate temporary relief, then aggravation of symptoms.	March 13, 1944. Carcinoma 3 by 3 cm. involving right arytenoid and ventricle.	Uneventful. Regained 60 pounds weight Returned to usual full time activity. Constructed artificial larynx for himself. Well and no evidence of recurrences 2 years after operation (Fig. 4a and b).
VII.	E.M. (339986) 66 years	Pain in region of larynx, 10 months. Dysphagia and hoarseness 7 months. January to March 1944 received 28 x-ray treatments and later 15 additional treatments. No relief.	Aug. 26, 1944. Carcinoma 4 cm. in diameter involving right posterior laryngeal aperture with extension on to hypopharynx and upper portion of cervical esophagus.	Uneventful. Increase of 8 pounds in weight. Returned to usual full time activity. Well and no evidence of recurrences 1 year 6 months after operation.
VIII.	H.M. (348593) 47 years	Hoarseness, 2 years. Received 40 x-ray treatments in 1942, with return of voice to normal quality. In October, 1944 laryngeal examination revealed recurrence of lesion.	Jan. 2, 1945. Carcinoma 3½ by 3 cm. at base of epiglottis and extending laterally on both sides to involve pyriform fossae.	Uneventful. Returned to usual full time activities. Well and no evidence of recurrences, 1 year 2 months after operation.
IX.	M.J. (349875) 59 years.	Hoarseness, cough, pain in throat, 8 months. Dyspnea 4 months. Dysphagia 2 weeks. Received 22 x-ray treatments during 6 months prior to admission without relief.	Feb. 8, 1945. Carcinoma 3 by 2 cm. involving both cords and producing marked constriction of aperture. Extensive fibrosis about upper portion of cervical esophagus. Several lymph nodes revealed metastatic carcinoma.	Uneventful. Because extensive resection of the cervical esophagus was necessary a gastrostomy was performed. Several attempts at plastic closure of esophagus were unsuccessful. One year after operation resection of metastatic mass high in left cervical region was carried out. This entailed resection of left common carotid artery; transient hemiplegia. Patient ambulatory and without gross evidence of recurrences 1 year 2 months after operation.
X.	F.W. (253647) 58 years	Hoarseness began in 1941. Biopsy in October 1941 highly suspicious of carcinoma. January 1945 received 5000 r unit x-ray therapy. Some improvement. March 1945 fistula developed over larynx, biopsy showed carcinoma.	May 21, 1945. Carcinoma involving much of larynx with fistula lined by carcinoma in anterior portion of neck. Cervical lymph nodes showed no metastases.	Uneventful. Living and well without recurrences 10 months after operation. Continuing his usual occupation.
XI.	A.G. (354516) 74 years	Hoarseness for 5 years. Recent difficulty in respiration. Tracheotomy March 1945. Laryngofissure right, in April 1945; sections showed carcinoma. X-ray therapy in May 1945. Progressive occlusion of larynx during summer 1945 due to recurrences.	October 3, 1945. Large carcinomatous mass involving most of larynx. Nodes contained no metastases.	Uneventful. Living and well without evidence of recurrences 5 months after operation.
XII.	M.G. (375872) 62 years	Hoarseness for 7 months. Dysphagia 2½ months. Finally almost complete obstruction necessitating tracheotomy 6 weeks before admission.	Feb. 16, 1946.	Uneventful. Recent case. Well, no evidence of recurrences 1 month after operation.

the latter. If cervical node metastases on one side are adherent to the jugular vein, the latter is also resected with them (Fig. 2). Resection of muscles or nodes piecemeal is to be avoided. At the close of the resection there is a large excavation in the neck, bounded laterally by the sternomastoid muscles, carotid arteries, and one or both jugular veins. Posteriorly, there is a strip consisting of the posterior walls of the esophagus and hypopharynx. Superiorly, there is the transected base of tongue or hyoid bone denuded of the muscles attached to its inferior border in the event that the hyoid is not resected. Inferiorly, there is the intact portion of the lowest segment of cervical esophagus, the remaining portions of thyroid gland and the transected trachea. The operation is terminated by passage of a Levine tube through the nose, pharynx, and well into the thoracic esophagus. The remaining portions of hypopharynx and cervical esophagus are closed over the tube by fine catgut sutures. The trachea is sutured to the skin margins of the inferior angle of the midline incision and the remainder of the wound is closed with interrupted silk; soft rubber drains are inserted to extrude from each angle of the horizontal segment of the incision which is made parallel to and slightly above the hyoid bone. A large

Fig. 2. Photograph of surgical specimen from Case IX showing *L*, larynx, with *C*, large carcinoma, and resected *M*, right sternomastoid muscle; *N*, right submaxillary gland and nodes; *N'*, right cervical nodes and *V*, right jugular vein.

size tracheotomy tube is inserted into the trachea. Nourishment is received through the Levine tube and consists of a gastrostomy diet of adequate calories, mineral, and vitamin content.

The large wound in the neck has usually separated to varying degrees during the second to third week after operation. A

TABLE II.—SUMMARY OF SURVIVAL IN 12 PATIENTS RECEIVING PANLARYNGECTOMY FOR ADVANCED LARYNGEAL CARCINOMA

SERIES A, 1939–1941
(Previously reported, 1)

Case	I.	Living and well—7 years. No recurrences.
Case	II.	Died 40 days after operation. Hemorrhage from tumor erosion of left carotid artery.
Case	III.	Survived 1 year. Died, recurrences.
Case	IV.	Survived 1 year, 7 months. Died of hemorrhage with recurrences. Received palliation.
Case	V.	Survived 6 months. Died of hemorrhage from recurrences.

Series B, 1943–1945

Case	VI.	Living, well, no recurrences. 2 years.
Case	VII.	Living, well, no recurrences. 1 year, 6 months.
Case	VIII.	Living, well, no recurrences. 1 year, 2 months.
Case	IX.	Living, comfortable. 1 year, 2 months. Recurrences present after 1 year.
Case	X.	Living, well, no recurrences. 10 months.
Case	XI.	Living, well, no recurrences, 5 months.
Case	XII.	Living, well, 1 month.

Fig. 3. Photograph of larynx from Case VI showing *C*, carcinoma, that was not controlled by previous irradiation therapy.

secondary repair of the esophageal fistula and wound in the neck is necessary. When this is finally accomplished the nasal tube is removed and a general diet is eaten in the usual manner. In the last 3 patients of this series healing almost per primam was obtained due, it is believed, to the use of penicillin systemi-

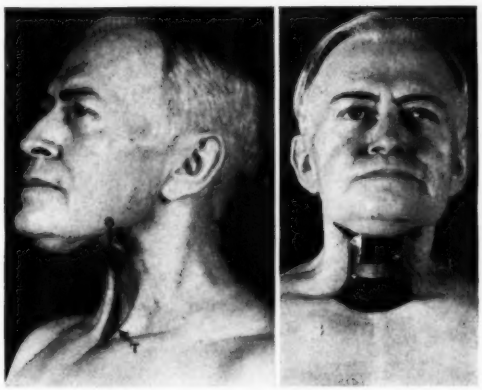

Fig. 4. a, left, Photograph of patient (Case VI) 2 years after panlaryngectomy showing "excavation" in anterior neck; *T*, opening of trachea above sternal notch with hard rubber tube inserted, and *O*, open sinus, into hypopharynx. The patient has gained 60 pounds by eating semisolid diet and holding small pad over the opening during feedings. He does not desire closure of the sinus since the present situation permits use of artificial larynx which he has constructed. b, Same patient with artificial larynx in place. This is readily placed and removed manually at will. Two years after operation patient is engaged in usual occupation requiring considerable physical and mental effort; no evidence of recurrences.

cally and the sucking of penicillin lozenges every 2 hours during the day for 2 to 3 weeks postoperatively.

Since the initial report mentioned, 7 additional patients have been subjected to this procedure. The first patient operated upon and cited in the previous report is living, well, without evidences of recurrences 7 years later and has returned to his regular occupation of farming during this period.

A summary of the additional 7 patients is presented in Table I; they are numbered serially, consecutive to the initial series of 5 patients previously reported. A recapitulation of the entire series (A and B) is presented in Table II.

The improved immediate results achieved in Series B, the second group of 7 patients, would appear to be fortuitous. The lesions presented were at least of the same magnitude as those in the first group. No selection of patients was carried out since the procedure was offered to all the patients referred with advanced laryngeal carcinoma, who presented no evidence of pulmonary or other distant metastases. There was no immediate surgical mortality in the series.

DISCUSSION

This report is prompted by the continued very pessimistic attitude generally existing in regard to advanced or extrinsic laryngeal carcinoma, especially when there have been recurrences following more conservative procedures and/or when there has been a failure of control following irradiation therapy. While the series of patients here considered is not a very large one it would appear that after failure of the more conservative operative procedures or irradiation therapy, there is still something to offer these patients if the process has not extended beyond the neck.

A larynx, the site of recurrent and advanced cancer, is the source of considerable distress which is often not greatly ameliorated by simple tracheotomy. Its wide resection constitutes a palliative measure, to say nothing of offering an opportunity for "cure," which experience has shown cannot be afforded by other means.

SUMMARY

Further experiences with panlaryngectomy have substantiated the view that the procedure is indicated in advanced carcinoma of the larynx and in recurrences following more conservative surgical procedures or irradiation therapy which has failed to control the process.

The procedure affords the opportunity for appreciable palliation and also for prolonged control of the disease (7 years in 1 case in the reported series).

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OPERATIVE TECHNIQUE OF THORACOLUMBAR SYMPATHECTOMY

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IN 1940 Smithwick (7) described the operation of thoracolumbar sympathectomy for the relief of hypertension. In several communications based on increasing experience with the operation, Smithwick, (8, 9, 10) has emphasized the value of the surgical approach in selected cases with definite improvement in 70 to 80 per cent of his patients. Ray and Hinton and Lord (1, 2, 3, 4) have confirmed Smithwick's results in smaller series of hypertensive patients. In view of the ever widening interest among internists and surgeons in the surgical treatment of hypertension, a description of a satisfactory operative technique employed on 227 patients may be of value.

Between February, 1942 and August, 1945, we removed the thoracolumbar sympathetic chain from the ninth thoracic to the second lumbar inclusive along with 2.5 inches of the greater splanchnic nerve and the lesser and least splanchnic nerves in 184 patients. From September to November, 1945, we extended the operation to include the sixth thoracic to the second lumbar inclusive and all of the greater splanchnic nerve in 28 patients. In December, 1945 and January, 1946, we removed the chain from the third thoracic or the fourth thoracic through the second lumbar in addition to the greater, lesser, and least splanchnic nerves *in toto* in 15 patients. The rationale for increasing the extensiveness of the procedure is based on the observation that the fall in the postoperative diastolic pressure is greater and seems to be maintained longer than in the less extensive procedure recommended by Smithwick. Whether the late results will show any greater benefit is entirely conjectural. In any case, the operation is of greater magnitude and hence is more serious for the patient. It is entirely possible that experience will show that the extensive two

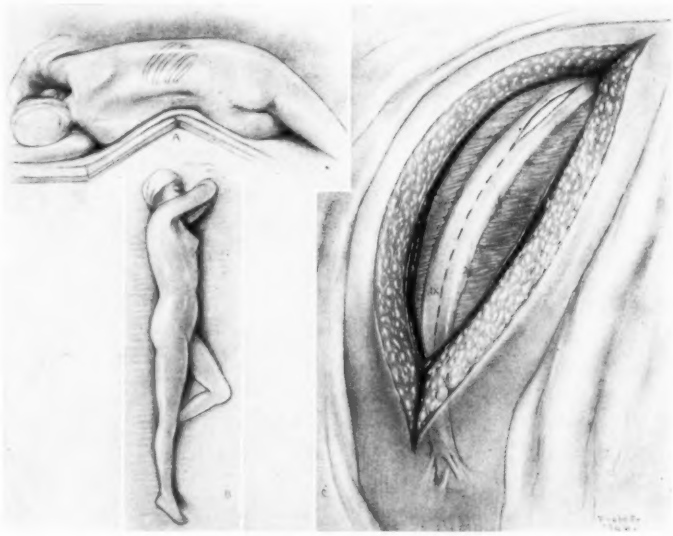

Fig. 1. A, B, Position of patient on table. C, Incision.

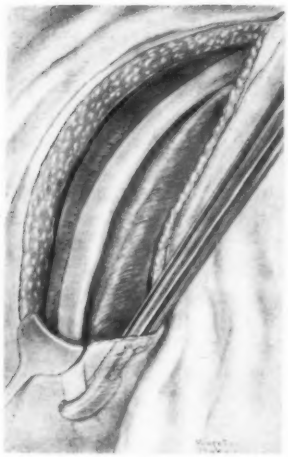

Fig. 2. Left, Drawing demonstrating removal of rib.
Fig. 3. The diaphragm is divided in its entirety on the side operated upon.

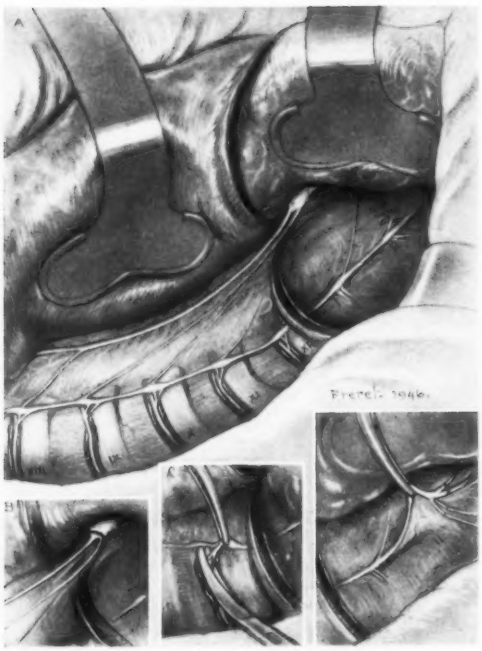

Fig. 4. Retraction of parietal pleura, lung, and retroperitoneal fat with large Harrington splanchnic retractors.

Fig. 5. Division is carried out above third splanchnic ganglion.

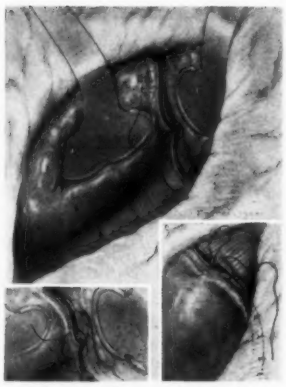

Fig. 6. Closure of wound.

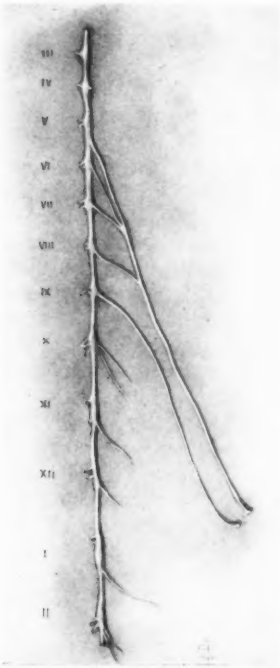

Fig. 7. Drawing of specimen removed *in toto*.

stage sympathectomy will be worthwhile only in a few selected patients.

Positioning. We place our patients in the exact lateral position with the lower leg flexed and the upper leg extended. This position is the one used by Dr. Carnes Weeks and he was the first surgeon in New York to do the thoracolumbar sympathectomy and had operated upon 40 patients before entering the service in January of 1942. The table is broken under the patient's lower costal margin (Fig. 1, A and B). If the twelfth rib is long then the tenth rib is resected subperiosteally in its entirety. If the twelfth rib is short the ninth rib is removed (Fig. 1, C and Fig. 2).

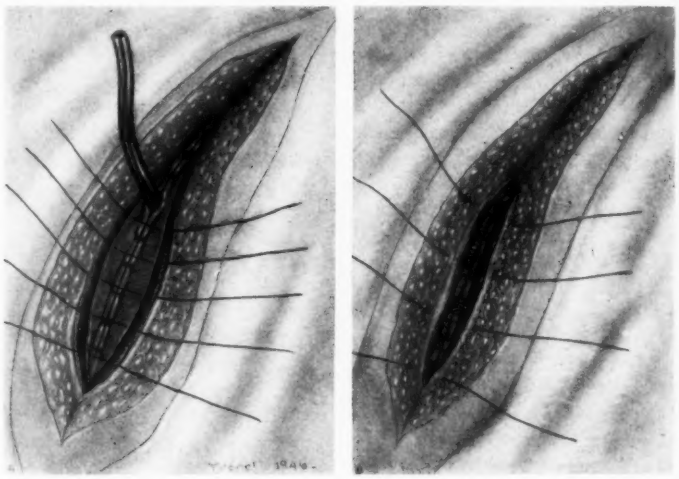

Fig. 8. Catheter is inserted into pleural space if pleura has been torn. The air is aspirated and catheter is removed at completion of muscle closure.

Following resection of the rib the parietal pleura is carefully reflected from the posterolateral chest wall exposing the diaphragm which is divided in its entirety on the operative side (Figs. 3 and 4, A). Retraction of the parietal pleura with contained lung and the retroperitoneal fat subdiaphragmically is facilitated by two large Harrington splanchnic retractors (Fig. 4, A). Delineation of the greater, lesser, and least splanchnic nerves along the lower thoracic and upper lumbar sympathetic chain is carried out without difficulty because of the wide exposure (Fig. 4, A). The greater splanchnic nerve is divided at its junction with the celiac ganglion (Fig. 4, B). The chain is grasped with a long curved hemostat and carefully dissected from each intercostal artery and vein (Fig. 4, C). The communicating rami are divided several millimeters from each ganglion (Fig. 4, C) and the chain divided just below the second lumbar ganglion (Fig. 4, D). The twelfth ganglion is usually located just above or in the substance of the diaphragm and we have repeatedly noted how attenuated the chain is between the twelfth thoracic ganglion and the first lumbar ganglion.

The thoracic chain is pursued cephalad until the third ganglion is mobilized and division is carried out above it (Fig. 5, B). In the majority of instances the greater splanchnic nerve has its origin from thoracic ganglia 6th, 7th, 8th, and 9th (Fig. 5, A). The lesser and least splanchnic nerves are removed as the thoracic chain is mobilized. Figure 7 is a drawing of a specimen removed *in toto*. One of the interesting findings in the operation of thoracolumbar sympathectomy is the wide variation in size and distribution of the nerves.

Closure is begun by joining the divided diaphragm with interrupted sutures (Fig. 6, A, B, C). If the pleura has been torn during the operation (and with this type of exposure it usually is opened) a rubber catheter is inserted into the pleural space and at the completion of the muscle closure, the air is aspirated and the catheter is removed (Fig. 8, A, B). Following this the skin is closed. We employ cotton as suture material throughout. As a rule intratracheal general anesthesia is used (5). The two stages are performed from 7 to 10 days apart depending on the condition of the patient following the first stage.

SUMMARY

An operative technique for the performance of thoracolumbar sympathectomy has been described. The ninth rib is usually resected thereby giving adequate exposure for the removal of the thoracolumbar chain from third thoracic to the second lumbar inclusive and also the entire greater, lesser and the least splanchnic nerves.

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CLINICAL EVALUATION OF SOTRADECOL, A SODIUM ALKYL SULFATE SOLUTION, IN THE INJECTION THERAPY OF VARICOSE VEINS

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A GREAT many types of solutions have been used for the injection therapy of varicose veins in the last few decades. Though they have been investigated pharmacologically the only definite conclusion which could be reached regarding their action is that they damage or destroy the intima of the veins and thereby produce a firm thrombus which after organization forms a fibrotic tissue resulting in permanent obliteration (5, 6). This has been achieved by the use of agents which obviously attack the tissues in different ways. For the discussion of the pharmacological action of these agents it is advisable to group them according to their probable pharmacodynamic action.

In the first group belong the agents whose action can be correlated with the physical properties of their solutions. To these belong (a) the *soap solutions* such as sodium morrhuate, sodium ricinoleate, sodium psylliate, monoethanolamine oleate, sodium linoleate, jecoroleate, and potassium oleate. These solutions are alike in that the soaps are present in them in the form of colloidal micelles, they are more or less alkaline, and they have a low surface tension. To this group belong also (b) *hypertonic solutions* of various crystalloid compounds, such as sodium chloride, calcium chloride, sodium citrate, dextrose, saccharose, invert sugar, and combinations of these substances. Their action is due to dehydration by osmosis.

In the second group belong substances which react chemically with components of the cells and tissues. To these belong (a) certain *heavy metals*, especially mercury bichloride, mercuric iodide, iron perchloride; (b) certain *organic compounds* which are known to react with proteins such as sodium salicylate quinine hydrochloride, and urethane; (c) compounds which contain *reactive halogens*, such as tincture of iodine, Lugol's solution, Pregl's iodine solution, and finally, (d) strongly *alkaline solutions*, such as sodium carbonate.

In recent years there has been considerably increased interest in the use of soap solutions for the injection treatment of varicose veins. Much of this interest is derived from the fact that the soaps are bland in action and do not cause violent reactions when accidentally injected into the perivenous tissues. Nor do they produce cramps and excessive pain which were often observed with the older sclerosing solutions, especially those belonging to the second group. These caused a rather diffuse tissue damage often extending beyond the walls of the veins and involving perivenous tissue. This action is also true of hypertonic solutions for, in order to cause sufficiently strong destructive damage to the intima, they have to be used in very highly concentrated solutions so that through diffusion of these diffusible substances damaging concentrations are built up throughout the entire vein wall and even in the adjacent tissues. However, the injection of soap solutions proved to be disappointing in that obliterations were not permanent and that certain other complications were noted (2, 9). These comprised serious allergic or anaphylactoid reactions ranging from simple skin eruption to shock and even sudden death. Unfortunately, the soap solutions are also notoriously nonuniform (4).

The possibility was conceived that the effectiveness and safety of sclerosing solutions could be increased by the use of substances whose surface activity is greater than that of soap solutions which are colloidal in nature and can be synthesized and obtained in a pure form. It was to be expected that the danger of the presence of allergens which are obvious-

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Fig. 1. Injections were made in duplicate with (*1*) 0.1 cubic centimeter 5 per cent soricin, (*2*) 0.1 cubic centimeter 5 per cent sotradecol, and (*3*) 0.1 cubic centimeter 5 per cent sodium morrhuate. Photograph was taken 24 hours later.

ly more liable to occur in organic substances of natural origin would be minimized with these agents. Various synthetic surface active agents of the alkyl sulfate and sulfonate type were tested and it was found that especially those which contain the highly branched chain alkyl residue are much more active in producing thrombosis and subsequent obliteration of veins in experimental animals than the soap solutions (8). The development of a bioassay, the tail vein of mice being used, was made possible through the production of a graded response with the variation of the concentration of the solutions. This procedure facilitated the exact comparison of various solutions. The solutions were tested for their irritating properties by subcutaneous injections in rabbits and were found to be almost entirely free from inflammation-producing properties. If they were injected intradermally they created a well circumscribed, strongly necrotic lesion, whereas soap-like agents under the same condition produced a necrosis with a rather wide edematous and erythematous halo. Results of such tests carried out on the shaved back of an albino rabbit in duplicate are shown in Figure 1.

From these results it appeared that the alkyl sulfates are more localized in their irritating action than are soap solutions. This may be due to the fact that they are practically neutral whereas soap solutions are usually quite alkaline.

Sodium tetradecyl sulfate was selected on the basis of this study as the most suitable alkyl sulfate for therapeutic use among those tested. Its toxicity was found to be of the same order of magnitude as that of the soap solutions (lethal dose 50=90 mgm. /kgm. intravenously in mice). Its activity is about two to four times as great. The solutions used in this study contained 1, 2, 3, and 5 per cent of sodium tetradecyl sulfate, 2 per cent benzyl alcohol, and were buffered at *p*H 7.6.

INDICATIONS AND TECHNIQUE

There are few therapeutic measures in which such a great variety of techniques are used as in the injection therapy of varicose veins. In order to enable the reader properly to evaluate the findings reported here we wish to discuss briefly the indications and technique used in this study.

In my opinion no sclerosing agent, no matter how great its safety and effectiveness, should be indiscriminately injected into varicose veins. As it was emphasized in a previous paper (1), and as it has been pointed out by many others, the Trendelenburg and Perthes tests have to be part of the proper general examination of the patient with varicose veins. The Trendelenburg test, if properly performed and interpreted, gives one precise information regarding the presence or absence of backflow or reverse flow from the deep veins into the superficial veins. Moreover, it is of value in estimating the degree of hydrostatic pressure within the vein in cases with demonstrable reverse flow. It is convenient to classify the patients in accordance with this test into four groups. Trendelenburg positive, Trendelenburg negative, doubly positive, and nil. The test is carried out as follows: The patient stands with both lower limbs exposed to the groin. All the superficial veins are inspected and palpated to ascertain their size and the

degree of tension or pressure within the vein. The patient is then placed in a recumbent position, the leg is elevated above the level of the pelvis and the veins are stroked or "milked" toward the groin thus emptying them. A tourniquet is placed around the upper thigh with sufficient pressure to block the superficial veins. The patient stands, and the veins below the tourniquet may remain collapsed and then fill slowly from below upward in 35 to 60 seconds, or longer in some cases. The test is repeated, and when the patient stands the tourniquet is released quickly. If the veins distend quickly from above downward to their former state of tension, the patient is classified as Trendelenburg-positive.

The Trendelenburg-positive group comprises those cases in which the valve function in the upper part of the great saphenous veins is deficient or absent, permitting reverse flow from the femoral vein into the saphenous vein at the saphenofemoral junction. This is the type of case most frequently encountered and it is most effectively treated by preliminary high ligation and division of the great saphenous vein, and separate ligation and division of all the tributaries at the fossa ovalis, with subsequent injection therapy.

The immediate benefits to be derived from a high ligation and division of the great saphenous vein can be demonstrated visibly by the Trendelenburg test; what the tourniquet accomplishes in preventing backflow in a valveless vein is precisely what the operation will do. So many surgeons in recent years have adopted this type of surgery that there can be little doubt of its efficacy.

According to deTakats (3) the pressure in a valveless great saphenous vein when the patient is standing may be as high as 210 centimeters of water. Obviously, high ligation as a preliminary measure in this type of case is the only means whereby this tremendous pressure or backflow can be prevented.

The *Trendelenburg-negative* case is one in which reverse flow from the deep veins into the superficial veins takes place through one or more dilated communicating veins, described by McPheeters as "blowouts." When the Trendelenburg test is performed in this group, the veins will fill rapidly when the patient stands with the tourniquet pressure maintained. If the varices in the leg are very large, preliminary ligation or ligations should be performed at the highest demonstrable point of backflow and all other points of reverse flow below it. In such cases a high ligation and division of the great saphenous vein is performed as an added measure of security. The sources of backflow from the deep veins may be more accurately ascertained by one of the modifications of the Trendelenburg test (7).

The *Trendelenburg-double* group comprises those cases in which the backflow takes place at the saphenofemoral junction as well as through one or more of the dilated communicating veins in the lower part of the limb. Briefly, it is a combination of the positive and negative types of backflow and the treatment obviously is high ligation and division of the great saphenous vein, and ligation or ligations in the lower part of the limb wherever backflow can be demonstrated. This group makes up only a small percentage of the cases encountered in practice. Pratt's method is ideal for localization of the incompetent perforating veins.

Trendelenburg nil cases are those with dilated veins in the presence of competent or normal valves. This is the early type of case frequently seen with no reverse flow. These varices may be obliterated by simple sclerosing injections.

In performing the Trendelenburg test, the status of the short saphenous vein must also be borne in mind. One frequently sees cases in which reverse or backflow takes place from the popliteal vein into the short saphenous vein at the popliteal fossa. In such cases the same principle of treatment is applied—namely, high ligation and division of the short saphenous vein at its junction with the popliteal vein.

The *Perthes test*, or Perthes' modification of the Trendelenburg test, is a simple test to determine patency of the deep veins as well as their functional capacity. Theoretically, it should be performed in every new case before treatment, operative or otherwise, is commenced. In the absence of any edema or previous thrombophlebitis, however, one may

proceed with treatment without performing the test. A tourniquet is applied above the knee with sufficient pressure to block the superficial veins only, while the patient stands and the veins are distended. The patient is instructed to walk about actively. If, with the tourniquet in place, the veins below it collapse (empty), then one may be certain that there is no serious deep vein blockage and the patient may be treated. On the other hand, if the veins become more tense and cause pain, deep venous occlusion must be suspected and conservative treatment should be given. This test may be performed with a tight elastic bandage applied to the limb to be tested from the toes to the knee or above it.

Recently several workers have reported treating varicosities of patients with a history of thrombophlebitis when the acute process had occurred a number of years previously and nature has had an opportunity to establish an adequate collateral circulation in the deep venous system. This attitude seems reasonable, since it is well understood that varicose veins are derelict veins, permitting reverse flow and in no conceivable manner functioning as normal veins in returning blood to the heart. The presence of edema and enlargement of a limb should make one suspicious of an insufficient deep vein circulation and great caution must be exercised before such patients are treated. In the absence of edema, however, it is safe to assume that the deep veins are patent and functioning well and to proceed with treatment in such cases. The constant application of the Perthes test, or one of its modifications, should give one experience in eliminating those cases not suitable for sclerosing therapy. In this connection the value of phlebography as diagnostic aid has been well established.

TECHNIQUE OF INJECTIONS

When high ligation and division were combined with injection therapy, injections were made soon after the operation. Large varices were injected with the patient in a recumbent position. The varices were emptied with the assistance of an occluder or isolated between tourniquets according to the method suggested by McPheeters. Small veins were often injected with the patient in an upright posture as this makes the veins more prominent and facilitates the procedure. Usually not more than two or three sites were injected at the same time in a distance of several inches. After injection a firm bandage was applied, especially when extensive portions of the vein have been treated. Usually 0.5 to 2 cubic centimeters of the solutions were injected at any one time and not more than 6 cubic centimeters of the 5 per cent solution, and 10 cubic centimeters of the 3 per cent solution were used. The 1 per cent solution was reserved for minor varices which are usually injected with a fraction of a cubic centimeter.

Altogether 327 patients were injected at the time when these results were compiled. These required a total of 2,064 injections, that is, an average of approximately 6 injections per patient. At first, patients were treated whose veins could not be obliterated with soap solutions. These cases were treated with the 5 per cent solution. It was soon realized that the 5 per cent solution, though well tolerated, is unnecessarily strong and I tried 3, 2, and 1 per cent solutions and found that for most purposes the 3 per cent solution was the most satisfactory.

RESULTS

The criteria used in judging the effectiveness of the drug were as follows: The extent, firmness of the thrombus, and the permanency of the obliteration were observed and compared with results obtained either on the same patient or on other patients with 5 per cent soap solutions. The incidence of recanalization was recorded up to the time of compilation of these data (September, 1945) which covered a period of 1 to 2 years for a good proportion of the patients. Among the side effects which often accompany sclerosing treatment the following were observed and recorded: incidence of sloughing, incidence of undue pain, development of cramps in the leg, incidence of discoloration and lumping (cosmetic results), and incidence of allergic phenomena such as skin eruptions or systemic reactions of the anaphylactoid type. These results are summarized in Table I.

Extent and firmness of obliteration. The first observations made with sotradecol were im-

pressive, as even in cases in which repeated failures were experienced with other sclerosing therapy, a firm, extensive thrombus was produced without any appreciable local reaction. It is believed that quite a few cases could have been treated with a smaller number of injections had an overcautious attitude not been necessary in view of the fact that this was the first experience with a new drug.

Incidence of failure in obliteration. Failure occurred in 5 cases, that is, 0.24 per cent. These failures were probably due to the cautious approach in the application of this new agent since on a second injection, in which somewhat larger doses were used the veins in all of these cases were obliterated in a satisfactory manner.

Recanalization. In only a fraction of the cases treated with sotradecol has there elapsed a sufficiently long period of time to allow the conclusion that recanalization has not occurred. In more than half the cases the time was not long enough to permit such conclusion. Therefore, in this communication the incidence of recanalization cannot be discussed statistically. However, it may be justifiable to state that so far no cases treated with sotradecol were seen which have recanalized, whereas observation for the same period of time of cases treated with soap solutions would have revealed a substantial percentage of recanalizations.

Sloughing. Sloughing occurred in only 4 instances (0.19 per cent) when veins were treated with a larger dose or a stronger solution than would have been indicated in view of the effectiveness of the agent. Two of these cases were superficial skyrocket type veins which were treated with 3 per cent sotradecol. It was later found that in the treatment of such cases 1 per cent solution is adequate and safe.

Pain. Pain occurred at the beginning when the 5 per cent solution was used more frequently than after the proper dosage had been established. It is felt that some degree of local reaction goes hand in hand with good and permanent obliteration and should not be regarded seriously. However, I have recorded all the complaints which appeared to be excessive, a total of 13 (0.63 per cent). Pain developed always several hours after injection and lasted 3 days in 1 case. Only 1 patient complained of burning sensation immediately after injection.

TABLE I.—SUMMARY OF RESULTS

Concentration—per cent	1	2	3	5	Total
Number of cases treated	33	80	173	207	493*
Number of injections made	105	313	813	833	2064
Failures in obliteration (1st injection)			2	3	5
Failures in obliteration (2nd injection)					0
Sloughing			4		4
Cramps			1	1	2
Pain	1	2	4	6	13
Pigmentation		1	1	2	4
Skin rashes			1		1
Systemic reactions			1	2	3

*The actual total number of patients treated was 327; a number of these were injected with more than one concentration of sotradecol, and thus, are represented more than once in the total.

Cramps. Cramps were observed in only 2 instances (0.1 per cent). They were of short duration and not nearly as severe as those experienced with other sclerosing agents.

Cosmetic results. The fibrotic tissue replacing the thrombotized vein was usually uniform and smooth. Lumps were not observed. Discoloration at the site of injection is not frequent. It occurred in 4 cases (0.19 per cent) and in 2 of these it persisted for several months. It is probably due to the fact that this compound, as well as the soap solutions, is hemolytic, and some of the liberated hemoglobin decomposes to hemochromogen and subsequently to hemosiderin which is resorbed only slowly. Discoloration can be considerably decreased by emptying the vein after the needle is inserted and before the injection is made.

Skin rashes. Skin rash developed after 1 injection (0.05 per cent). The same patient tolerated subsequent injections well and without symptoms. It may be noteworthy that complaints of itching were made in 6 additional cases but did not recur since the practice was adopted of dressing the site of injection with a dry, sterile, cotton pledget instead of one moistened with alcohol. All the reactions which might have been of allergic nature occurred after the first injection with sotradecol. It is, therefore, believed that the reaction is not due to anaphylaxis and that allergy is not de-

veloped during the treatment with sotradecol. Reaction is more likely to be connected with its lytic, cell-destructive nature.

Systemic reactions. The incidence of these was 3 cases (0.15 per cent). Two of these complained of chills a few hours after the injection. Both of these patients were injected subsequently with the same solution without producing any side effects. One patient had an anaphylactoid attack at the time of the first injection with sotradecol. He had reacted similarly to soap injections so that the injection treatment of this patient had to be discontinued.

CONCLUSION

The sclerosing therapy of varices has its limitations. One reason why it has recently fallen in disrepute in the minds of some surgeons is that the agents used were not adequate and that they had to be used in ineffective doses to avoid grave sequelae. The experience with sotradecol reported above indicates that a substantial improvement of the sclerosing therapy has been made possible with this new agent. The sodium salt of tetradecyl sulfate apparently has great advantages over the sclerosing solutions used previously inasmuch as it is much more potent, relatively less toxic, and produces a less diffuse and more localized reaction. It is not entirely free from producing untoward side effects though in this respect, too, it is far superior to the soap-type sclerosing agents.

SUMMARY

Sodium tetradecyl sulfate solutions were tested on 327 cases by 2,064 injections for their therapeutic efficiency in the treatment of varicose veins with and without ligation of the saphenous vein and tributaries. They were found to be two to four times more effective than were soap solutions. Their action appeared to be stronger and less diffuse. Side effects such as pain, cramps, sloughing, skin eruptions, and systemic reactions occurred much less frequently than with other sclerosing agents. The pharmacology of this compound was discussed in comparison with agents used previously.

NOTE.—Since the compilation of this report several hundred additional injections were made with essentially identical results. Untoward side effects have been almost completely eliminated as more experience has been gained regarding the proper dosage.

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THERAPY FOR SOFT TISSUE SARCOMAS

An Experimental Study

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THE choice of treatment of soft tissue sarcomas is one of the most trying problems for both the radiologist and the surgeon since the tumor responds poorly to radiation therapy and often recurs following surgical excision. Local recurrences are often ascribed to the surgeon's failure to demand radical excision, and the radiologist may serve to defer more extensive surgical procedures by expectantly radiating the tumor. Hence physicians often apply several types of therapy. A search of the recent literature reveals little clinical or experimental data upon which one might critically evaluate the methods employed.

Our present knowledge, gained principally from the clinical studies (7) indicates that most fibrosarcomas recur locally following excision and often metastasize, after local recurrences, to the lungs. The rate of growth and the rapidity of recurrence (1) of a sarcoma were roughly proportional to the degree of malignancy as judged microscopically. Too, it has been noted that wide excision of the neoplasm or amputation if the mass is located on an extremity serves to forestall metastases.

The classification of sarcomas of soft tissues is not satisfactory to most morphologists. Ewing regards most of the malignant tumors arising in soft tissues as neurosarcomas; others (8) fail to accept this concept and hold that the production of collagen indicates the tumor to be one developing from fibroblasts, if the nerves present are not forming a portion of the neoplasm.

Undoubtedly the bulk of malignant tumors classified as sarcomas of soft tissues originate from fibroblasts, produce collagen, and the nerves present do not form an integral part of the new growth; thus they may be better classified as fibrosarcomas.

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Physicians are often perplexed when confronted with a tumor which has been designated a fibrosarcoma. Amputation can readily be recommended if a massive tumor can be found on an extremity, but he will hesitate to apply this procedure for smaller lesions that might be checked by it. Again the clinician may defer drastic measures for a small or seemingly localized tumor if the pathologist's report indicates the tumor to be a "low grade malignancy."

To solve the dilemma there is a growing tendency to apply radiation therapy in varying quantities to soft tissue sarcomas found in most sites. The radiologist, having been encouraged by his colleagues, who are dismayed at radical procedures, administers palliative irradiation which seldom produces serious disability and usually satisfies all concerned that the diseased part is receiving treatment. Perhaps due in part to the different opinions held as to the classification of soft tissue sarcomas and perhaps due to the sparsity of suitable data regarding the efficacy of various types of treatment, there is at present no uniform opinion held concerning the most suitable methods of treatment (4, 5).

In order better to evaluate these methods, it would seem desirable to study the effects of each type of therapy applied singly to the same neoplasm. This is made possible by the use of transplanted animal tumors. By employing spontaneous or experimentally induced neoplasms from homogeneous inbred animals, fragments of the same tumor may give rise to any desired number of uniform growths in other animals of the same inbred stock. These transplanted neoplasms, with one hundred per cent predictability of progressive growth, may be treated in an environment as similar to that of the primary host as the identical twin of the primary cancer patient. The purpose of the present paper is to

TABLE I.—NUMBER OF TREATED AND UNTREATED RATS OF EACH EXPERIMENT, THE NUMBER WITH METASTASES, AND THE AVERAGE SURVIVAL PERIOD OF THOSE DEAD WITH TUMORS, AND THE PER CENT OF CURES

Experiment	Tumor	Experimental procedure	No. of rats	No. dead with tumor	Average no. of days survived	Tumor weight in grams	Gross metastases		Microscopic lung metastases	Per cent cures
							Lymph node	Lung		
I	4337 148A	Surgical excision after 17 days	9	9	40	29	4	0	5	0
		Untreated	10	10	31	36	6	2	7	0
II	4337 151A	Surgical excision after 10 days	10	10	40	32	8	2	10	0
		1000 r units after 10 days	10	10	39	20	9	4	10	0
		Untreated	10	10	34	16	7	0	9	0
III	4337 153A	Amputated after 7 days	10	5	63	0	5	3	3	50
		1500 r units after 7 days	9	9	50	32	9	7	8	0
		Untreated	10	10	33	16	5	3	8	0
IV	4337 159B	Amputated 7 days 1500 r units on 7th and 9th days	7	2	61	0	2	2	2	71
		1500 r units on 7th, 9th, and 11th days	10	7	67	29	7	7	7	30
		Untreated	11	11	30	14	8	2	7	0

report the results obtained with such a tumor by several surgical and radiation techniques.

MATERIAL

Induced rat sarcoma 4337 was the neoplasm selected for these experiments. The primary tumor, composed of bundles and whorls of uniformly large, plump spindle cells, was induced by Cysticercus fasciolaris from presumably normal cells residing in the liver cyst wall. *In vitro* studies (6) and a history (2) of its growth on transplantation have been previously published. When transplanted to the subcutaneous tissues, it grows rapidly, becoming palpable in about 7 days, and invariably causes the death of the host in 3 to 7 weeks.

EXPERIMENTAL PROCEDURE

At the beginning of the present experiments the tumor was in the 148th transplanted generation. Uniform grafts weighing approximately 2 milligrams were implanted in the subcutaneous tissues of the dorsal side of the right forefoot of the same inbred line as the host of the primary tumor. Surgical or radiation therapy was instituted after 7, 10, or 17 days and an equal number of inoculated rats for each experiment were maintained untreated as controls. At necropsy the size and weight of the tumor mass was recorded and the superficial lymph nodes and lungs were examined for metastases.

The roentgen technique used was as follows: 100 kilovolts, 4 millimeters of aluminum filtration, 15 milliamperes and 25 centimeters skin target distance. The field size depends on the size of the lesion, but was never more than 5 centimeters in diameter. In experiment II, the rats were irradiated while under anesthesia. In all subsequent experiments a lead lined box was used to immobilize the rats. A hole of sufficient size to withdraw the affected limb was made in one side of the container. The latter method facilitated the treatment of the lesion and excluded the remainder of the body from the primary beam of the x-ray as well as much of the scattered irradiation. Too, it made possible the fractionated treatment without repeated insults from anesthetics.

RESULTS

The results are summarized briefly in Table I and shown graphically in Figure 1. In the first experiment the tumors were allowed to grow untreated for 17 days. At this time they averaged 3.0 by 2.0 by 1.5 centimeters in size and weighed approximately 2 grams when completely excised. No attempt was made to

remove a margin of uninvolved tissue but the entire mass of the tumor was removed, the skin drawn over the area operated upon, and the opening closed with interrupted silk sutures. One rat was accidentally overanesthetized but the 9 which survived all had recurrent tumors. The average survival period was 40 days compared with 31 for the controls. The tumors averaged 29 grams in weight at death compared with 36 grams for the controls. Gross superficial lymph node involvement was observed in 4 of the rats operated upon and 6 of the control rats. None of the rats operated upon and 2 of the controls showed gross lung metastases but microscopic foci were found in the lungs of 5 of those operated upon and 7 of the control rats. The difference between the two groups was not very striking. The average difference in survival period of 9 days in favor of the group operated upon would be equivalent to nearly a year in a man's life and the resultant tumor masses were somewhat smaller in the group operated upon.

In experiment II, the tumors of one group were surgically removed after 10 days and in a similar group were irradiated at the same time, receiving 1000 roentgen units of x-ray. The results were very similar to the observations in experiment I. In the case of the group operated upon all of the tumors recurred and at the death of the host had a greater average weight than either the tumors of the control or the irradiated group. The average survival period for the controls was 34 days and 39 and 40 days, respectively, for the irradiated and operated upon groups. All but 1 of the controls showed microscopic foci in the lungs. Two of those operated upon and 4 of the irradiated rats showed gross lung involvement. The superficial lymph nodes showed microscopic involvement in 8 of the operated upon, 9 of the irradiated, and 7 of the controls.

In experiment III, the right foreleg was amputated 7 days after inoculation from each of the rats of one group and a similar group received at the same time a treatment of 1500 roentgen units of x-ray on the right forefoot. As a result of amputation at this time 5 or 50 per cent of the rats operated upon were completely cured. They were killed after 8

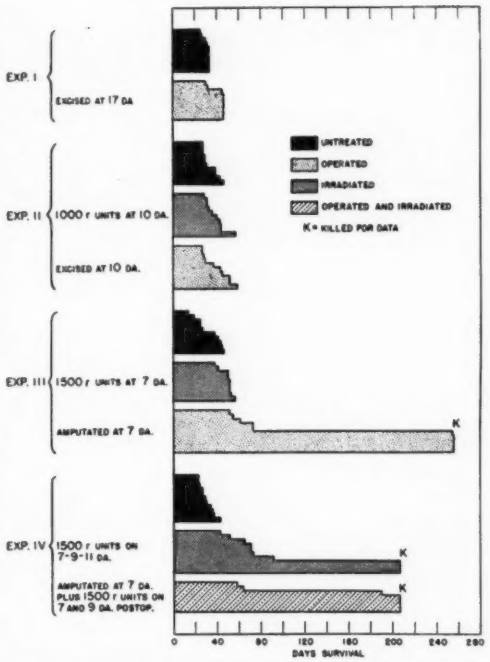

Fig. 1. The survival period of each rat of the several experiments.

months and a thorough postmortem examination revealed no gross involvement; too, no foci of involvement were discovered by microscopic examination of the lungs, superficial lymph nodes, and stump. In the case of the other 5 operated upon rats, the stump healed completely and showed no involvement. They all outlived the controls with an average survival period of 63 days compared with 33 days for the untreated. At necropsy all showed gross involvement of the superficial lymph nodes of both sides and had lung metastases from foci which must have been established within the 7 days prior to the operation.

In experiment IV, the therapeutic measures were also instituted on the 7th day after inoculation. In the case of the group operated upon the right foreleg was amputated and on the 7th and 9th succeeding day each was given 1500 roentgen units of x-ray on the healed stump and the axillary lymph node region of the right side. The irradiated group were each given a course of fractionated treatments

of 1500 r units each on the 7th, 9th, and 11th day after inoculation. The controls all died with tumors in an average of 30 days, 8 of the 11 had gross axillary lymph node metastases and 7 also showed lung involvement. Seven of the 10 rats which received 4500 r units of x-ray died with large tumors in an average of 67 days. One died on the same day as the last of the untreated rats and the others outlived them by a considerable period. Three were apparently cured. The 7 which died with tumors all had gross lymph node and lung involvement but the 3 apparent cures were killed after 207 days and showed no trace of the tumor at the site of inoculation on the foot or in the lungs and lymph nodes. Following the treatment in each case, the hair on the treated foot fell out, the skin became reddened, eventually broke down and was covered by a crusty scab. The treated feet of the 3 cures eventually healed but were deformed. The growth of the primary tumor in each of the other 7 was considerably retarded.

Only 7 of the group operated upon survived the experimental procedure, 3 having succumbed to an overdose of nembutal. Five were completely cured, and 2 died with extensive lymph node and lung involvement. The 5 apparent cures were killed after 207 days and no trace of the neoplasm was found in the lungs, lymph nodes, or stump. There were too few rats in this group to be sure that these results were any better than those obtained for the operated upon rats of the previous experiment in which amputation was followed by no further treatment. Irradiation of the lymph nodes failed to control the growth of metastases in 2 of the treated rats. There is no doubt, however, that the larger, protracted, dose of x-ray gave better results than the single radiation treatment of the 2 previous experiments. Thirty per cent were completely cured and the others survived an average of more than 30 days longer than the controls, an increase in survival period equivalent to more than 3 years of a man's life.

SUMMARY

1. Rats bearing a transplanted rapidly growing spindle cell sarcoma (Cysticercus induced sarcoma 4337) on the right forefoot received surgical and irradiation techniques.

2. Complete surgical excision 10 or 17 days after inoculation was followed by a recurrence of the neoplasm in all cases but average survival period was increased 6 to 9 days.

3. Amputation of the afflicted limb 7 days after inoculation completely cured 50 per cent of the tumor bearing rats and prolonged the average survival period of the others which died from previously established lung or lymph node metastases by 30 days, an equivalent of 3 years of a man's life.

4. Local irradiation with 1000 or 1500 roentgen units of x-ray 10 and 7 days, respectively, after inoculation prolonged the average survival period 5 and 17 days beyond that of the untreated controls but effected no cures.

5. Irradiation with 4500 roentgen units (a course of treatment consisting of 1500 r units given on the 7th, 9th, and 11th day after inoculation) caused complete cures of the tumor in 30 per cent of the treated rats and prolonged the average survival period of the remainder by more than 30 days.

6. Local irradiation with 3000 roentgen units (1500 r units on the 7th and 9th days) after amputation of the afflicted limb failed to control the growth of metastases established in the lymph nodes prior to amputation in 2 of 7 rats.

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TRANSTHORACIC OPERATIONS FOR NEOPLASMS OF THE ESOPHAGUS AND STOMACH

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DURING the past ten years a number of surgeons have developed a more or less standardized procedure for resecting carcinomas of the lower end of the esophagus and cardiac end of the stomach, with re-establishment of direct continuity between the esophagus and the remaining portion of the stomach. The idea that the surgical treatment of the thoracic esophagus and cardia of the stomach are impractical and excessively dangerous procedures has been challenged successfully. The old idea that the blood supply of the esophagus is insufficient to support the healing process of an anastomosis has been ruled out, and the walls of the esophagus have been found sturdy enough to hold a suture line. Contributing to the success of these procedures are the modern methods of intratracheal anesthesia and the use of penicillin and the sulfa drugs. The surgical approach is through the thorax to the abdominal cavity, which is opened by incising the diaphragm at the esophageal hiatus. This approach is also used in the treatment of benign lesions of the lower esophagus and stomach.

In presenting a small group of patients on whom we have used the transthoracic approach in operating on both benign and malignant lesions of the lower esophagus and upper stomach, we realize that a number of surgeons have far greater experience in this procedure than we have. It is their work which has made it possible for the general surgeon successfully to approach this problem. Increasing reports of single cases or small series of cases reported in the literature indicate that the general surgeon has entered this field, and it is our opinion that the operation has not yet reached the stage at which the report of a few additional cases is without further value.

From the Department of Surgery, Stanford University Medical School.

Read before the meeting of The Western Surgical Association, Chicago, 1945.

This report deals with our experience with this operation at Stanford University Hospital. Sixteen patients have been operated upon; in 7 patients with carcinoma a successful resection and anastomosis was made. Five patients had inoperable lesions; but by freeing the diaphragm around the growth, these patients were able to eat following operation, which they had been unable to do previously. One supected case of carcinoma which proved to be inflammatory was resected. This patient died of a cardiovascular accident the day after operation and was the only hospital death in the series. In 3 patients with megaesophagus an esophagogastrostomy was successfully performed.

HISTORY

The present method of transthoracic approach for lesions of the esophagus and stomach has been the outgrowth of years of effort on the part of many surgeons and one in which development of thoracic surgery has played a dominant rôle. The importance of this subject is emphasized by the frequency with which carcinoma of the esophagus occurs. Souttar gives its incidence of all malignant disease as from 4 to 6 per cent, and Ernst at from 6 to 10 per cent. San Francisco statistics would indicate it to be on the increase; its incidence to all malignancies was 4.4 per cent from 1920 to 1923, 5.9 per cent from 1924 to 1927, and 6.1 per cent from 1928 to 1931. Regarding its location in the esophagus, Janeway and Green found that almost 85 per cent of the lesions occurred in the middle and lower thirds of the esophagus.

The almost total failure of radiation therapy in carcinoma of the esophagus leaves surgery as the only present hope of treatment. Against the obvious surgical difficulties of their location is the fact that many of these lesions are slow to metastasize and slow in their extension locally. Our present discussion

Fig. 1. Exposure through left interspace and crushing of phrenic nerve.

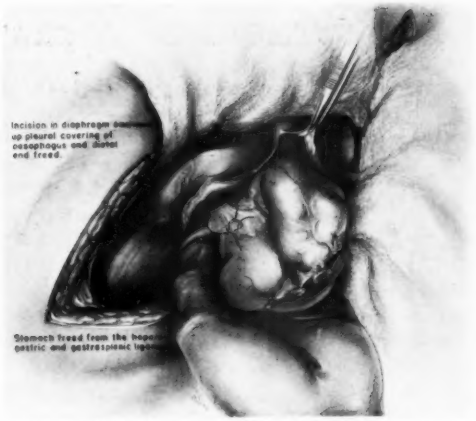

Fig. 2. Diaphragm open and mobilization of stomach carried out.

deals only with cancer low in the esophagus and the cardia, so we shall not go into the manifold problems and attempted solutions presented by cancer higher in the esophagus.

In 1871 Billroth first demonstrated in animal experiments that resection of the esophagus was feasible. In 1905 Sauerbruch performed experiments on cadavers, which consisted of transthoracic mobilization of the lower end of the esophagus and its invagination through the cardia into the stomach. At a second stage, through laparotomy, the stomach was opened and the invaginated portion of the esophagus removed. Ohsawa reported using this procedure in 2 cases, 1 of which was successful.

Using the abdominal approach, Voelker in 1907 and Kuemmell in 1909 resected carcinomas of the cardia and performed esophagogastrostomy. Henle, reported by Tiegel, was one of the first to use the thoracic abdominal approach. A laparotomy was first done and the stomach severed below the tumor. Esophagogastrostomy was done with a Murphy button. The patient died of collapse of the left lung. In 1937 Adams and Phemister were the first to operate successfully in one stage transthoracically, resecting a carcinoma of the lower esophagus and doing an esophagogastrostomy. It is their technique which we have followed. Ochsner and DeBakey in 1938 successfully removed a carcinoma of the cardia with esophagogastrostomy, performing the operation in two stages; laparotomy first and thoracotomy second.

Garlock (7, 8, 9) has been able to resect a lesion and make his anastomosis cephalad to the aortic arch. He reports doing a Torek type of operation in 15 cases and an intrathoracic anastomosis in 14 cases. Thirteen patients died, an operative mortality of 44.8 per cent. During the past 4½ years, he has had 14 operable cases with 5 deaths, an operative mortality of 35.7 per cent. Of the 9 surviving patients, 1 is well almost 4 years; 1, 18 months; 1, 13 months; 1, 7 months; and 1, 6 weeks. Four patients died of local recurrences or other cause; 1 in 22 months; 2 in 1 year and 1 died 3½ months after operation.

Stephens reports 23 cases of cancer of the thoracic esophagus and upper end of the stomach that underwent surgical exploration. Ten of these involved the lower end of the esophagus and cardiac end of the stomach. Resection and reanastomosis was carried out in 5 instances. A similar procedure was done in 2 cases in which the tumor involved the esophagus only, and in a footnote he adds 3 more cases, making a total of 10 cases up to 1941 in which resection and direct anastomosis was done. Twenty-two months after resection only 1 patient remained alive of the original 23.

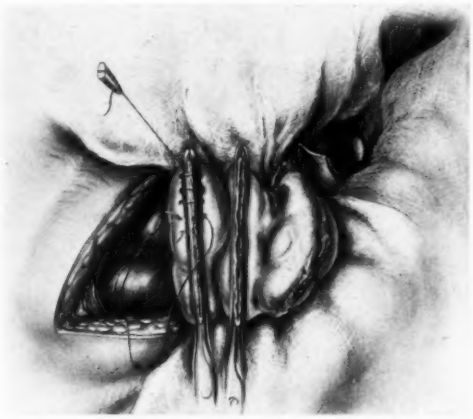

Fig. 3. Resection of stomach and exposure of esophagus carried out.

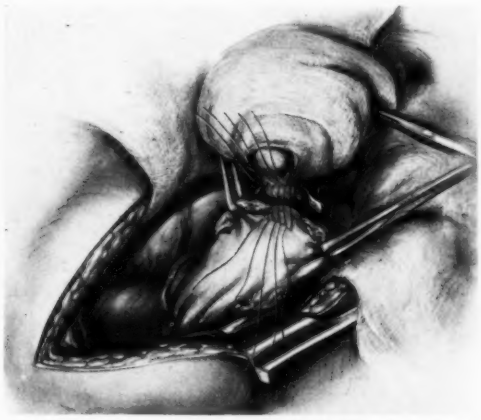

Fig. 4. Placing posterior row of sutures between stomach and esophagus.

Phemister reports a patient with an esophagogastrostomy alive and well 3⅓ years after operation. Sweet has recently reviewed 127 cases in which the transthoracic approach was used at the Massachusetts General Hospital. In 85 patients the lesion was in the lower esophagus or stomach. In 61 patients a resection was done; 24 cases were inoperable. The mortality was 19.5 per cent; of the patients who survived, only 3 are alive and well 3 or more years after operation. Nevertheless, Sweet states that if after radical surgery the patient is relieved of his obstruction and can swallow again satisfactorily, if only for 6 months to 1 year, the operation should be considered worth while. Among other contributors in this field are Kay, Dorsey, and Churchill.

PREOPERATIVE CARE

The patients are usually elderly and in varying stages of starvation, making adequate preoperative preparation an essential for success. Preparation of our patients for surgery consisted of hydration of the patient with intravenous glucose and saline until a satisfactory urinary output was attained. Following this the packed cell volume of the whole blood and plasma protein were determined, and if anemia or hypoproteinemia existed, these conditions were corrected by transfusions of red blood cell concentrates or by whole blood. The esophagus was washed out daily; and 24 hours prior to operation nothing further was allowed by mouth, and an indwelling Levine tube was placed with continuous Wangensteen suction. The suction was maintained through the surgical procedure. Appropriate doses of crude liver extract, vitamin C, and B complex vitamins were given intramuscularly daily from admission to discharge. Twenty-four hours before surgery 5 grams of sodium sulfadiazine were given intravenously and 15,000 units of penicillin were administered intramuscularly every three hours.

ANESTHESIA

Intratracheal anesthesia was used in all of our patients. The anesthetic agent varied. The early patients were anesthetized with cyclopropane and oxygen or with ether and oxygen. Later cases in the series received ether and air as described by Neff and Mayer of our department of anesthesia.

OPERATIVE PROCEDURE

With the patient lying on the right side, the left pleural cavity is opened in the eighth or ninth interspace. At first we resected the ninth rib but have found this unnecessary. Inspection and palpation of the esophagus furnishes evidence as to the operability of that portion of the lesion lying above the

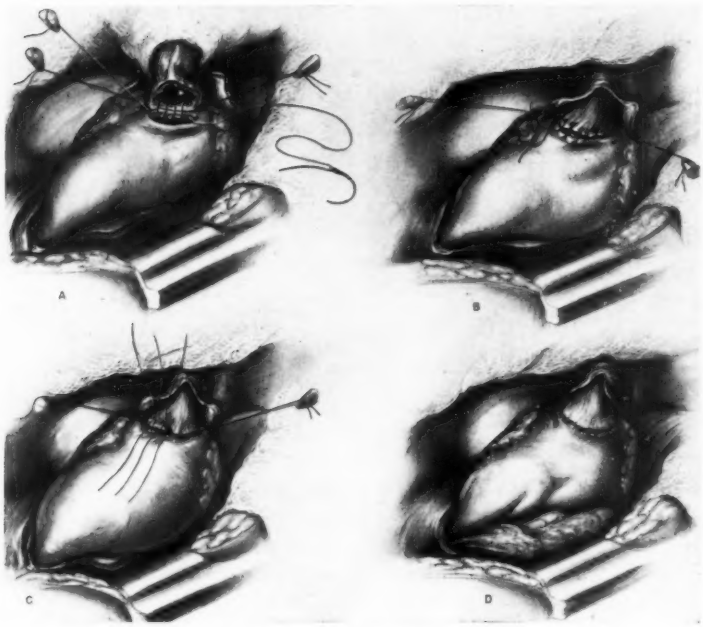

Fig. 5. Completing the anastomosis.

diaphragm. If the outlook is favorable, the phrenic nerve is pinched to paralyze temporarily the diaphragm and the tendinous portion of the latter is incised above the esophagus. Even in those lesions which finally proved to be inoperable, considerable dissection and freeing of tissues were done before their inoperability was acknowledged. In the operable cases the initial step is to free the stomach and that portion of the esophagus which is to be removed. One of the technically important steps in the operation is adequate mobilization of the stomach and, if necessary, the duodenum. This is essential so that the distal portion of the stomach can be pulled up into the thorax without tension. One of the first to point out the importance of this procedure in relation to lesions of the lower esophagus was Brun in 1913.

Splenectomy was performed in cases in which tumor extensions made this necessary. The stomach is divided distal to the tumor and the end turned in with a Parker-Kerr stitch of chromic catgut reinforced with interrupted silk sutures. The proximal segment containing the lesion is turned upward for traction on the esophagus while the anastomosis is started. The anastomosis is made on the most available portion of the remaining stomach at the point where it seems to lie most naturally with a minimum of distortion and no tension. This point is usually on the anterior wall of the stomach. The esophagus is divided half through at the level selected for anastomosis above the lesion, and a posterior row of interrupted silk mattress sutures is placed and tied before the anterior half of the esophagus is divided. All anastomoses were done by the open technique, by using a mucosal row of continuous No. 00 chromic catgut and two rows of interrupted silk sutures. Before closing the anastomosis, the Levine tube is passed well into the stomach. An anterior row of interrupted silk sutures completes the anastomosis.

In all of our patients the blood supply of the esophagus appeared to be adequate. In order to avoid any unnecessary trauma to the esoph

agus, no clamps are used on it. The anastomosis is placed above the diaphragm and the cut edges of the diaphragm are sutured to the gastric wall with interrupted silk sutures in such manner as to relieve tension on the line of anastomosis. We allow the mediastinal pleura over the esophagus to remain open over the suture line to provide free drainage into the pleural cavity if a leak should occur, as we feel that an empyema could be treated, while a mediastinitis could not. Before closing the thorax, 50,000 units of penicillin dissolved in 50 cubic centimeters of saline is poured into the pleural cavity and a small mushroom catheter inserted in the tenth interspace in the posterior axillary line for underwater drainage. The lung is then expanded and the chest closed in layers with silk. During the procedure, the patients are supported with whole blood transfusions, plasma, and saline as indicated, the chief reliance being placed on whole blood. Patients are returned to the ward with continuous nasal oxygen, which is maintained as long as indicated.

POSTOPERATIVE CARE

In general the patients did well after operation. Measures required to fortify the patient's resistance were continued. Sulfadiazine and penicillin were given as long as appeared necessary. The intercostal mushroom catheters ceased to drain after 48 hours and were removed. Wangensteen suction was maintained through the Levine tube, which was removed about the fifth day. Our idea in continuing suction for this period was to prevent mechanical strain on the suture line, which would result from vomiting. On the day the tube was removed, an ounce of water was given each hour. Each day the quantity was doubled until 4 ounces were taken hourly; then the patient was started on small feedings of soft foods given 6 times daily. Later the diet was slowly increased to normal. The patients got up on the fourth or fifth day.

POSTOPERATIVE COMPLICATIONS

Prompt recognition of complications is essential. Our first patient did not receive penicillin therapy, and although he was dismissed in good condition on the twelfth postoperative day, he returned after 1 week complaining of a pain in his chest. A week later an x-ray film revealed a pleural effusion and he was readmitted for daily aspiration of the fluid and penicillin therapy. The fluid was found to be sterile. He was dismissed but returned a week later with shortness of breath. He was tapped again and pus was aspirated. A rib resection and tube drainage of the empyema were carried out and the empyema rapidly cleared up.

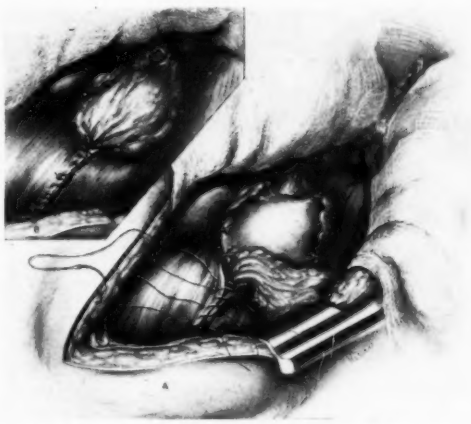

Fig. 6. Completion of anastomosis, closure of diaphragm, and suture to anchor stomach to diaphragm.

Another lesson was learned from this patient. He found that he would regurgitate after eating if he lay down after meals. X-ray studies with barium revealed no obstruction, so we advised him to remain upright for an hour after eating. He had no further trouble. We then advised our other patients to follow this procedure. It seems that when these patients lie down after eating, there is no sphincter action of the cardia and the gastric contents flow easily into the upper esophagus. One other patient developed a sterile pleural effusion which was cured by one aspiration. There has been only 1 death in this series of patients. In that case, the patient died of a coronary attack on the day after operation.

SUMMARY AND CONCLUSIONS

1. Thirteen patients were operated upon through a transthoracic approach for lesions of the lower esophagus and cardiac end of the stomach.

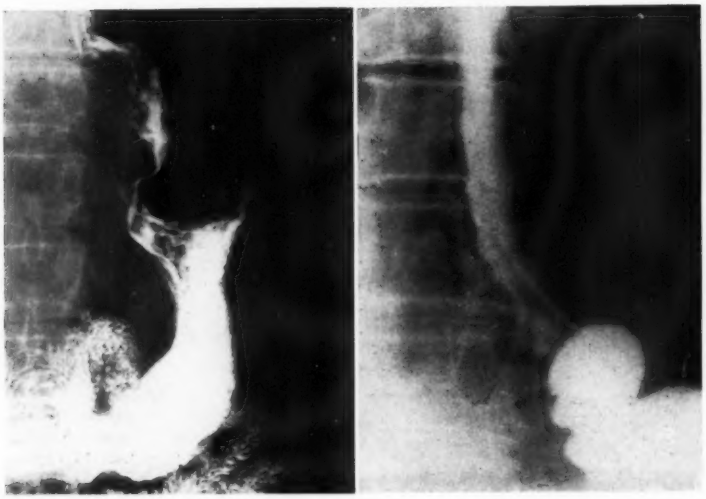

Fig. 7. Left, Case 5. Carcinoma of cardia of stomach extending into esophagus.
Fig. 8. Case 5. Postoperative view showing anastomosis.

2. Resection of the lower esophagus and portions of the stomach were done in 7 patients for carcinoma.

3. Resection was done in 1 case of suspected carcinoma which proved to be inflammatory. In this case the patient died on the second postoperative day of a cardiovascular accident. This was the only hospital death in the series.

4. Five patients with carcinoma were explored and found to be inoperable. In 1 patient, the spleen was removed; and in all, a varying degree of dissection was done in an attempt to free the growth before its inoperability was acknowledged. All of these patients were temporarily relieved of their dysphagia. In 1 case this relief lasted for 9 months.

5. Of the 7 patients in whom a carcinomatous lesion was resected, 1 patient after temporary relief died in 9 months of metastases. Another was known to have metastases 6 months after operation and has not been heard from; this patient is presumed dead. Four patients are well from 6 months to over a year after operation. One man 11 months following operation has gained 31 pounds, eats regularly, and feels fine. Another patient is still convalescent in the hospital.

6. On the basis of these few cases and reports in the literature, one may cautiously say that resection of carcinomatous lesions of the lower esophagus and stomach offers a slight hope and a measure of relief to a group of previously hopeless cases.

CASE REPORTS

CASE 1. R. W., No. 232642, male, aged 46 years, negro, entered the hospital June 26, 1944, complaining of vomiting after taking food and vomiting large amounts of saliva between meals for the past 6 months. He had lost 30 pounds. X-ray studies revealed a normal esophagus with stasis at the cardia of the stomach. The fundus contained a ragged, neoplastic lesion. The pylorus and duodenum were normal.

Operation: On July 12, 1944, the chest was entered through the bed of the ninth rib. Upon opening the diaphragm, a large tumor of the cardia and fundus was found. It grew almost to the cardioesophageal junction and extended over half of the stomach, distally. The regional glands along the greater curvature were involved. The lesion was resected, including a 3 centimeter margin of uninvolved tissue, distally, and 2 centimeters of the esophagus. The end of the stomach was closed with a layer of No. 1 chromic catgut and two layers of silk mattress sutures. The esophagus was anastomosed to a separate opening on the anterior surface of the stomach. Sulfanilamide crystals were dusted about the anastomosis and the diaphragm and chest were closed. A catheter was inserted for drainage.

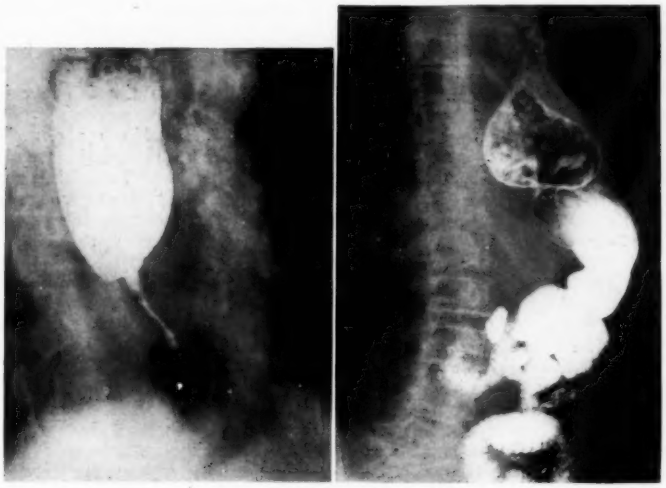

Fig. 9. Left, Case 6. Carcinoma of lower esophagus.
Fig. 10. Case 6. Postoperative view showing supradiaphragmatic portion of stomach and anastomosis.

Subsequent course: The intercostal catheter was removed after 48 hours, the Levine stomach tube was removed on the fifth postoperative day. He was dismissed on July 25, 1944. One week after dismissal he developed a pleural effusion, followed 1 week later by empyema. The empyema cleared up with open drainage and penicillin therapy.

This patient was last seen March 22, 1945, when barium studies were done. There was very little regurgitation into the esophagus in either the prone or supine position. The supradiaphragmatic portion of the stomach appeared smaller than on previous examinations. There was no spasm at the hiatus. The duodenum was normal. He felt well and ate a normal diet.

Microscopic diagnosis: Adenocarcinoma of the stomach.

CASE 2. J. A., No. 210087, male, aged 69 years, white, entered the hospital August 15, 1944, complaining of difficulty in swallowing for 1 month. Esophagoscopy revealed a fungating, ulcerated mass occluding the lumen 40 centimeters down the esophagus. Barium studies showed a freely moving esophagus in the region of the tumor. Biopsy revealed a squamous cell carcinoma.

On August 15, 1944, the chest was opened through the bed of the ninth rib and the distal esophagus explored. A firm, nodular thickening was found involving the distal 9 centimeters of the esophagus and extending 3 to 4 centimeters into the cardia of the stomach. The diaphragm was opened anteriorly and a resectable lesion was found. The esophagus and upper third of the stomach were mobilized and the tumor was resected with a margin of uninvolved tissue above and below the lesion. The patient had been prepared for operation with penicillin and before closing the chest, 50,000 units of penicillin were poured into the chest about the anastomosis.

The recovery was uneventful. He was dismissed September 10, 1944, in good condition and eating a soft diet. He has been followed in the out-patient clinic and when last seen, November, 1945, he was having no difficulty referable to his operation and was eating a normal diet. He has had periodic X-ray studies with barium and his anastomosis has functioned well. There have been no signs of recurrence of his tumor.

CASE 3. S. P., No. 235067, male, aged 52 years, negro, entered the hospital October 1, 1944, complaining of vomiting after eating any appreciable amount of food. On one occasion he vomited material containing flecks of blood. He had lost 45 pounds. There was a palpable, nontender mass in his left upper epigastrium. Barium studies showed delay at the cardia and the upper half of the stomach was rigidly constricted into a narrow tube. The diagnosis made was neoplasm of the stomach, probably of the linitis plastica type.

On December 4, 1944, the ninth rib was removed and the chest explored. The esophagus was normal, but the upper half of the stomach was a thickened, nodular tube. Extensions of the tumor were found in the gastrosplenic ligament and in the region of the tail of the pancreas. A splenectomy and partial resection of the tail of the pancreas were necessary. After resection of the lesion, only one-third of the stomach remained. It was necessary to mobilize the first and second portions of the duodenum in order to approximate the end of the esophagus without undue tension. Before closing the chest a drain was

passed through the left lateral abdominal wall down to the region of the resected tail of the pancreas. After closing the diaphragm, 50,000 units of penicillin were placed in the chest and the chest was closed. A catheter drain was inserted through the tenth intercostal space.

On the first postoperative day the patient's temperature rose to 38.9, but returned to normal on the second day and remained normal thereafter. He was dismissed October 21, 1944, in good condition. He was followed in the out-patient clinic and got along well for 6 months and then began to lose weight. A palpable recurrence developed in his left upper abdomen. He left this locality at that time and has not been heard from since.

CASE 4. E. G., No. E41402, male, aged 69 years, white, entered the hospital October 10, 1944, complaining of difficulty in swallowing for 2 months. Barium studies revealed a neoplastic lesion of the cardia of the stomach, which extended over most of the stomach and involved the lower 3 centimeters of the esophagus. Diagnosis was neoplasm of the stomach.

On October 12, 1944, the chest was opened through the bed of the ninth rib, explored and the diaphragm opened. The whole stomach was involved with tumor and there was a direct extension to the liver edge and a projection to the aorta. An attempt was made to mobilize the lesion by removing the spleen. By mobilizing the pancreas, an attempt was made to bring up a loop of jejunum to the esophagus to shunt by the stomach. This procedure was impossible so the case was declared inoperable and the diaphragm and chest were closed.

The patient was relieved by the operation and was able to eat a soft diet up until about 1 week before his death which occurred about 4 months after operation.

CASE 5. E. R., No. E42470, female, aged 55 years, white, entered the hospital December 15, 1944, complaining of difficulty in swallowing and indigestion for 4 months. She said that food seemed to stick in her chest, causing severe pain, and for the past month liquids had the same effect. She had lost 20 pounds. Barium studies revealed a neoplastic lesion involving the cardia of the stomach up to the esophagus. Diagnosis was neoplasm of the cardia of the stomach.

On December 22, 1944, the chest was entered through the bed of the ninth rib. Upon opening the diaphragm, the tumor was found involving the cardia and lower 2.5 centimeters of the esophagus. The lesion was resected, including a 2.5 centimeters margin of uninvolved tissue on either side of it. The remainder of the stomach and the esophagus were anastomosed.

On the fourth postoperative day the patient complained of a tight feeling in her chest; on the sixth day, an x-ray was taken revealing a pleural effusion. Aspiration removed 800 cubic centimeters of clear, sterile fluid. The effusion did not reform. The remainder of her course in the hospital was uneventful and she was dismissed January 8, 1945. She lived comfortably and was able to eat satisfactorily until her death 9 months later. Her death was due to distant metastasis not involving the site of operation.

Pathological examination revealed squamous cell carcinoma.

CASE 6. F. L., No. 43515, male, aged 72 years, white, entered the hospital December 18, 1944, complaining of occasional vomiting for 8 months. Food seemed to stick in his esophagus but by pressing on his throat he could stimulate vomiting and gain relief. As this condition progressed he had the sensation that gas on his stomach prevented him from swallowing. Belching gave temporary relief and allowed food to pass for a while, but for 1 month before entry he was able to take only liquids. He had no pain. He had lost 50 pounds. Doctors who had seen him previously were unable to demonstrate an organic lesion by esophagoscopy. After admission esophagoscopy showed a neoplastic stenosis of the lower esophagus and marked dilation above the lesion. Biopsy revealed a squamous cell carcinoma.

On January 5, 1945, the chest was explored and the lesion found in the esophagus about 2 centimeters above the cardia extending upward for 4 centimeters. The lesion was resected, removing 3 to 4 centimeters of the cardia. The end of the esophagus was anastomosed to the stomach.

The patient was dismissed January 24, 1945, after an uneventful recovery. Ten months later he was seen in the out-patient clinic. He was eating well and had gained 30 pounds.

CASE 7. P. F., No. 238497, male, aged 62 years, white, entered the hospital January 25, 1945, complaining of vomiting and weight loss for 2½ months. Food seemed to stick in his throat, followed by a gurgling sensation which was relieved by vomiting. He had lost 37 pounds. Barium studies revealed a mass constricting the lower esophagus and filling the cardia of the stomach. Esophagoscopy showed a lesion constricting the esophagus beginning at a level 33 centimeters from the upper incisor teeth. Biopsy revealed a squamous cell carcinoma.

On February 7, 1945, the chest was explored and a tumor involving the lower 8 centimeters of the esophagus and extending into the cardia was found. The mass was invading the liver by direct extension and secondary nodules of metastases. The case was declared inoperable and the chest was closed.

The patient was relieved by the operation. He could eat a soft diet and was dismissed February 16, 1945, but died 1 month later.

CASE 8. J. S., No. 239676, male, aged 55 years, white, entered the hospital March 7, 1945, complaining of difficulty in swallowing for 6 months. He first noticed that pieces of meat stuck in his throat and soon started vomiting all solid food. For 1 month before entry he could swallow only liquids. He had lost 25 pounds. X-ray examination revealed a lesion of the esophagus at the level of the aortic arch. Esophagoscopy and biopsy showed a squamous cell carcinoma.

On March 12, 1945, the chest was explored and the lesion partially mobilized. It was found strongly adherent to the posterior aspect of the aorta. The case was inoperable and closed.

The patient felt relieved and was able to eat a soft diet. He was dismissed the sixteenth day. We have no further follow-up.

CASE 9. M. R., No. 240308, female, aged 64 years, white, entered the hospital April 1, 1945, complaining of difficulty in swallowing for 2 months. At first pieces of meat seemed to stick in her throat, causing pain which was relieved by vomiting. Later she had the same trouble with soft foods. She had lost 15 pounds. Barium studies showed a constricting lesion of the esophagus at the level of the aortic arch. Esophagoscopy revealed a neoplastic lesion constricting the esophagus 23 centimeters from the upper incisor teeth. Biopsy showed a squamous cell carcinoma.

On April 6, 1945, the chest was opened, the esophagus explored and a hard, nodular tumor was found at the level of the arch of the aorta. It was fixed to the posteromedial aspect of the aorta and invading the lung. The case was inoperable and closed.

On the day after operation the patient was able to swallow fluids and later able to eat soft foods without great difficulty. She was dismissed April 16, 1945. Follow-up letters have not been answered.

CASE 10. F. D., female, aged 70 years, white, entered the hospital April 12, 1945, complaining of difficulty in swallowing for 4 months. She had been on a liquid diet for 1 month. Esophagoscopy revealed stenosis at the lower end of the esophagus. A thread was passed in preparation for a dilator but when a French olive bougie No. 41 was passed, it met a very hard stricture at the cardia. Biopsy was done but the tissue obtained was insufficient to make a diagnosis. Without further attempts to make an exact diagnosis, surgery was advised.

On April 25, 1945, the chest was explored. A thickened, firm mass was found in the esophagus projecting through the esophageal hiatus and extending into the posteromedial aspect of the cardia. It gave the appearance and feel of an inflammatory mass. The lower esophagus and upper stomach were mobilized. The stomach was resected 8 centimeters below the cardia and the esophagus divided 4 centimeters above the mass, and an anastomosis made.

The patient got along well until the day after operation when she suddenly developed an acute cardiac attack and died within a few minutes. Postmortem examination was not obtained. Pathological examination of the resected specimen showed stenosis of the esophagus by periesophageal abscess tissue. Multiple, small abscess cavities containing pus with gram positive bacilli were found.

CASE 11. J. C., No. 239806, male, aged 43 years, white, entered the hospital May 13, 1945, complaining of weakness, tiredness, and some epigastric pain for 1 year. On occasions he had vomited watery fluid which contained streaks of blood. He had lost 30 pounds. X-ray examination revealed a rigid, irregular thickening of the fundus of the stomach, extending down the lesser curvature almost to the incisura. The duodenum and esophagus were normal. Diagnosis was carcinoma of the stomach.

On May 17, 1945, the chest was entered through the eighth intercostal space and the diaphragm opened. A palpable mass was found in the stomach wall on the lesser curvature extending from the cardia to within 8 centimeters of the pyloric ring. There seemed to be no regional spread of the tumor. The distal esophagus, stomach, duodenum were mobilized and the lesion was resected at the end of the esophagus and at a level about 3 centimeters proximal to the pyloric ring. By completely mobilizing the duodenum it was possible to approximate the esophagus to a point on the prepyloric region of the stomach. The common bile duct was not placed under undue tension. The Levine tube was passed well into the duodenum and the diaphragm and chest were closed. Fifty thousand units of penicillin were placed in the chest and a catheter drain inserted through the ninth interspace.

On the first postoperative day the patient's temperature rose to 39.8 and he continued to run a fever. On the third day, he was found, by chest x-ray examination, to have developed a bronchopneumonia on the right side. With continued penicillin and sulfa drug therapy his temperature returned to normal and on the seventh postoperative day his chest was cleared. On the ninth day a soft diet was started and the remainder of his course was uneventful. He was dismissed June 2, 1945. Since dismissal he has gotten along well, gained weight, and his only complaint is that he could not eat enough at one time to satisfy his appetite. He was last seen on August 27, 1945. X-ray examination showed a well functioning stoma at the anastomosis.

CASE 12. F. K., male, aged 59 years, white, entered the hospital July 29, 1945, complaining of difficulty in swallowing for 5 months. This difficulty had progressed to a point where was able to take only small amounts of liquids. X-ray examination showed a neoplastic lesion of the cardia end of the stomach with obstruction of the esophagus. Esophagoscopy showed an annular constriction of the lower esophagus. Biopsy revealed a squamous cell carcinoma.

The chest was entered on August 2, 1945. The tumor of the stomach could be felt through the diaphragm. It was large, firm, and fixed. An opening was made in the diaphragm in attempt to free the tumor but the local involvement was so extensive that, after some effort, the attempt was given up and the chest closed.

The recovery was uneventful. The patient was able to swallow with greater ease and take semisolid food. Nine months after the operation he died.

CASE 13. J. S., No. 246701, male, aged 63 years, white, entered the hospital October 26, 1945, complaining of difficulty in swallowing and burning epigastric pain for 2½ months. He had no vomiting but was unable to eat solid food during this time

and had lost 30 pounds. X-ray studies revealed a filling defect in the lower esophagus just above the cardia. Esophagoscopy demonstrated a firm, partially constricting lesion low in the esophagus. Biopsy showed a squamous cell carcinoma.

On November 3, 1945, the chest was explored through the eighth interspace, revealing a 3 centimeter mass about 2 centimeters above the diaphragm. The esophagus and upper stomach were mobilized and the lesion resected, leaving a 4 centimeter margin of uninvolved tissue on either side. The stomach was anastomosed to the esophagus and the diaphragm closed about the stomach. Fifty thousand units of penicillin were poured into the chest and a catheter drain placed in the ninth interspace.

The patient's recovery was uneventful. The chest catheter was removed after 48 hours; the Levine tube was removed on the fifth postoperative day. He was dismissed on November 14, 1945, in good condition and eating a soft diet without difficulty.

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VASCULAR INJURIES IN WAR

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THE observations recorded in this paper were made in an evacuation hospital during the recent European war. In scope, they are limited to injuries of the major vessels of the extremities.

During the early months of our war experience our attention was drawn to this group of casualties by the high incidence of circulatory gangrene following primary ligation. Surgeons in other units with whom we discussed the subject reported results as discouraging as our own.

The realization that many of these ischemic extremities would go on to gangrene and amputation if nothing more active than ligation was undertaken led some surgeons to suture lacerations and bridge defects with glass or plastic tubes.

Among the 16,000 surgical admissions handled by our evacuation hospital unit there were 110, or 0.7 per cent, with injuries of the major peripheral arteries. There were 4, or 3.6 per cent, deaths in this vascular group. Amputations were done in 46, or 42 per cent, of the group, 39 for circulatory gangrene, and 7 for gas bacillus infections superimposed on ischemic extremities. Twenty-five per cent of our gas bacillus infections occurred in this group of casualties.

In no instance was amputation undertaken as a primary procedure. After shock control these casualties were sent directly to the operating room. It was felt that early operation gave the best chance of saving these extremities. When the vessels were only ligated, release of tension improved whatever circulation existed. Parenthetically, we might say that recognizable arteriovenous fistulas with small penetrating wounds should not be handled in this manner. Ligation in such cases may precipitate circulatory gangrene. Only 2 arteriovenous fistulas were recognized in our hospital, but, in all likelihood, others became clinically evident later.

It must be understood that in a front line hospital saving the patient's life is the prime consideration. Many of the patients with vascular injuries had other major associated injuries which precluded extensive vascular repair. Again, many of these extremities appeared to be in an irreversible state on admission. There were times when as many as 300 patients were in the preoperative wards awaiting surgery. These facts will, in part, explain our high percentage of simple ligation following débridement. Active retrograde bleeding gave a good prognosis and in such cases simple ligation was the procedure of choice. Pertinent information is lacking on some cases seen early in the campaign. Later, a careful study was attempted in all vascular injuries.

OPERATIVE PROCEDURES

Primary ligation was carried out in 88 cases, suture in 4 cases, suture anastomosis in 2 cases, glass or plastic tube anastomosis in 5 cases, débridement in 6 cases. There were 5 cases in which no operation was undertaken.

Ligation. Arteries that had not been completely severed were divided before ligation. With few exceptions, chiefly in the arm, companion veins were divided and ligated. In many instances they also had been injured.

Of 88 injuries so treated amputation was subsequently carried out in 27 or 30.6 per cent. Included in this group are 3 cases in which elective ligations were done at a point just distal to a large collateral vessel as well as at the point of injury. This procedure was recommended by Holman who has shown that the elimination of a blind, pulsating segment increases the systolic pressure in the collateral branches.

Suture. Three of 4 sutured arteries were brachials and the extremities were viable when the casualties were evacuated. In the fourth case gangrene appeared despite arter-

iotomy, removal of thrombus, and repair of a contused femoral artery. The common carotid artery was sutured in 2 instances without complications but these cases are not included in this series.

Suture anastomosis. Severed axillary and brachial arteries were anastomosed, and the casualties evacuated with intact extremities on the sixth and eleventh postoperative days, respectively. Unfortunately, the clinical status prior to operation was not recorded.

Glass or plastic tube anastomosis. Glass or plastic tubing was used to bridge arterial defects in 5 cases. Two casualties, one with a severed femoral and the other with a severed popliteal artery, were evacuated with apparently viable extremities on the sixth and thirteenth postoperative days, respectively. A popliteal anastomosis and a brachial anastomosis were unsuccessful and the extremities were amputated. In the fifth case, a popliteal injury, an extensive resection of anemic leg muscles was done. The outcome was so doubtful that it has been included among the amputees for statistical purposes.

Débridement. In 6 patients treated by débridement, 2 amputations were carried out. Four patients with femoral injuries in which the extremities were clinically viable prior to operation were evacuated with intact extremities. A cold extremity with a contused thrombosed popliteal artery progressed to gangrene and amputation. An extremity containing a thrombosed, lacerated brachial artery was also amputated.

No operation. Of the 5 patients in the group not operated upon, 2 died in shock shortly after admission. In a third, with a closed popliteal injury due to a displaced fracture amputation was carried out. A patient with an arteriovenous fistula of the femoral artery was evacuated with a warm leg. A casualty with a small perforating wound of the thigh had the peroneal extensor muscle group of his leg excised before evacuation. On admission the foot was warm, although no pulse could be palpated.

POSTOPERATIVE CARE

Following surgery patients with vascular injuries were sent to an especially designated ward. Further observations were recorded on a mimeographed record by the surgeon assigned to the ward.

Measures to promote development of collateral circulation were instituted. Smoking was not permitted. Whiskey at 6 hour intervals was encouraged. Despite the availability of a good brand of bourbon this therapeutic endeavor was not universally acclaimed by the patients. The injured extremities were kept at a level approximately 6 inches below that of the heart and left exposed to room temperatures.

Sympathetic blocks were done at 12 hour intervals in patients who exhibited signs of circulatory inadequacy. They were discontinued when the circulation appeared adequate or when there was a repeated lack of response.

In the lumbar regions the first and second lumbars were blocked with 30 cubic centimeters of 1 per cent novocain. When the arteries of the upper extremity were injured the stellate ganglion was blocked through an anterior approach with 15 cubic centimeters of novocain.

It did not seem to us that paravertebral blocks were of great value in the treatment of these casualties. When soft part damage is extensive, collateral vessels are destroyed. When the main vessel is interrupted, sympathetic blocks do not aid the flow of blood to the periphery. In a large number of cases a definite increase in skin temperature could be elicited, but we did not feel that the same response occurred in the more important underlying muscles.

One might wonder why certain extremities were not amputated primarily if the continuity of the vessel was not restored. It was difficult to be sure of one's clinical impression in many instances and we hoped blocks would benefit these casualties. We were also anxious to preserve as much extremity as possible, and as we were able to keep these men under observation, awaited evidence of demarcation.

Hematocrit and plasma protein determinations were done at frequent intervals. In an effort to restore blood volume, large quantities of blood and plasma were used. Penicillin and sulfadiazine were used in every case.

While questionable extremities were under observation there was very little elevation of temperature even among those that came to amputation. This fact can no doubt be attributed to chemotherapy.

Ischemic extremities going on to gangrene were invariably painful. Tachycardia and an anemia that showed little response to transfusion were the most consistent evidences of toxemia.

The duration of stay in our hospital was variable. Many patients with obviously viable extremities were evacuated within a day or two of operation. Early evacuation was at times necessary because of the tactical situation. Self-addressed follow-up cards accompanied all patients with vascular injuries. Unfortunately, all were not returned. As a rule, patients were held until either viability was established or amputation undertaken. We felt that casualties in whom the circulation was questionable were "nontransportable."

In those patients without nerve injury, the most reliable prognostic sign was the presence or absence of function. Functioning muscles have an adequate blood supply. Increasing edema made the prognosis poor. We were reluctant to elevate these extremities and incisions made to release tension were of no apparent value. In 4 cases of warm, edematous extremities in which incisions were made, avascular muscle was found. The anterior peroneal muscle group showed the highest percentage of avascular muscle.

AMPUTATIONS

All amputations were of the circular type and skin traction was applied at the time of operation. If infection was present, the traction was deferred for 2 or 3 days.

In accord with established policy, amputations were done at the lowest possible level. In many patients with associated fractures, this meant leaving a distal free fragment. We have often speculated as to the usefulness of these stumps. Follow-ups have been lacking so no definite conclusion can be drawn.

The discrepancy between the levels of viable muscles and viable skin was striking. In those instances in which amputation was done below the site of injury, viable muscle was encountered several centimeters proximal to the level of viable skin.

Of 170 major amputations performed by the unit, only 43 were done for gangrene following vascular injuries, the remainder as a result of mutilation of the extremity. In the latter, amputation was done as a primary procedure.

RESULTS

TABLE I.—GENERAL RESULTS

Artery	No.	Viable No.	Viable Per cent	Amputees No.	Amputees Per cent	Gas infections* No.	Gas infections* Per cent	Deaths No.	Deaths Per cent
Femoral	31	15	48	14	45	3	10	2	6
Popliteal	24	6	25	17	71	1		2	
Anterior and posterior tibials	8	1		7		2		0	
Subclavian	1	1							
Axillary	6	3		3		1		0	
Brachial	40	35	87	5	12	0		0	
	110	61	55	46	42	7	6	4	4

*Are included in the amputee column.

The average periods of observation were: femoral 5 days, popliteal 5, anterior and posterior tibials 6, subclavian 9, axillary 7, and brachial 4 days.

FEMORAL ARTERY

There were 31 femoral injuries. Of these 15 or 48 per cent were considered viable at the time of evacuation which averaged 5 days postoperative in this group. Amputation was necessary in 14 patients or 45 per cent of the series; gas bacillus infections occurred in 3 and amputation was necessary in each instance. There were 2 deaths. One patient had a partial traumatic amputation through the distal third of the leg and failed to respond to shock therapy. The second died in shock 5 hours after ligation of a severed artery.

It is significant that fractures were associated with 9 of the 14 patients in whom amputation was done and with only 4 of the 15 who left with intact extremities. There was, likewise, a predominance of severe soft part wounds in the former group.

TABLE II.—SITE OF INJURY AND THE OUTCOME IN FEMORAL ARTERY INJURIES

Artery	No.	Viable	Amputee No.	Amputee Per cent
Common femoral	2	0	2	
Superficial femoral	27	12	13	48
Unspecified	2	2	0	

The time interval between injury and operation averaged 24 hours with extremes of 5 and 72 hours.

TABLE III.—OPERATIVE PROCEDURES IN FEMORAL ARTERY INJURIES

Operation	No.	Viable	Amputees	Deaths
Débridement	3	3		
Débridement and ligation (sec.)	1	1		
Ligation artery	1	1		
Ligation artery and vein	20	6	13	1
Elective ligation	1	1		
Suture	1	0	1	
Tube anastomosis	1	1		
No operation	3	2		1

Of the débridement group of 3 cases in 2, the surgeons elected not to disturb the vessels as bleeding had ceased. No. 6487: A severed artery was present, and the patient was evacuated on the third postoperative day with a warm foot. No. 7428: A perivascular hematoma was encountered in a cool extremity. At evacuation on the fifth postoperative day the extremity was considered viable. In the third case, No. 12504, with a warm leg, an arteriovenous fistula was recognized after the débridement had been started. The procedure was terminated, and he was evacuated without complications.

The one patient, No. 11281, in the débridement and secondary ligation group had a 3 day old, infected wound which was débrided on admission. Secondary hemorrhage occurred 4 days later and was controlled by local ligation of a lacerated artery. The extremity was warm on admission and remained so despite the ligation.

In patient No. 11665, a severed artery in a warm extremity was ligated and a small vein laceration repaired.

In one patient, No. 14755, of the elective ligation group, it was noted that prior to operation the extremity was warm with a weak posterior tibial pulse. A lacerated artery and vein were ligated in the midthigh 23 hours after injury. There was no bleeding from the distal segment. Following the suggestion of Holman the superficial femoral was ligated just distal to the profunda femoris in order to eliminate the blind distal segment and to increase the systolic pressure in the profunda. When the casualty was evacuated on the fourth postoperative day his leg was warm and functioning. In view of the palpable pulse preoperatively, it is difficult to understand why retrograde bleeding was not obtained unless the surgeon failed to dislodge all the clot from the distal end of the vessel.

Suture was done in 1 patient, No. 14706. A contused, nonpulsating artery was incised 15 hours after injury and a clot was removed. After repair of the arteriotomy; the circulation was not maintained and gangrene followed.

In patient No. 13140, operation was begun 11 hours after injury in a cool extremity with absent dorsalis pedis and posterior tibial pulsations. A glass tube was inserted in a 4.5 centimeter defect of the artery in the midthigh, active retrograde bleeding was present, and an intact femoral vein was preserved. On evacuation 6 days after operation the extremity was cool but function was good.

No operation was performed in 3 cases. No. 4207 failed to respond to shock therapy on admission. No. 7256, a clinically recognizable arteriovenous fistula in a small perforating wound of midthigh, was watched for 3 days and then evacuated with a warm leg and palpable pulse. No. 14764 had a small perforating wound in the proximal third of the thigh and a warm, pulseless foot. Because of edema, incisions were made in the lateral aspects of the calf 5 days after injury. The extensor muscles of the leg were found to be avascular and were excised. The flexor group of muscles was intact when he was evacuated 10 days after injury.

POPLITEAL ARTERY

There were a total of 24 patients with popliteal injuries. Of these, 6 were evacuated with intact extremities and were observed for an average of 5 days. In 17, 71 per cent, amputation was carried out. One amputation was done for a gas bacillus infection in an ischemic extremity. There were 2 deaths; a casualty admitted in irreversible shock was not operated upon. The second died of a toxic nephrosis 3 days after amputation.

Fractures were associated with 10 of the 15 cases that came to amputation and with only 1 of the viable extremities. Soft part damage was described as severe in 13 of the amputations.

The average interval from injury to operation was 20 hours in 22 cases with extremes of 3 and 72 hours.

Ligation was done in 2 cases. Two of 4 viable extremities had adequate collateral circulations on admission. There is no preoperative description of the

other 2 but it is presumed that they did also. On the other hand, the amputees on whom there is a preoperative description invariably had avascular extremities before operation.

Elective ligation was carried out in 2 cases. No. 11666: On admission the leg was cool and pulseless. Operation was started 5 hours after injury. Soft part damage was slight. A lacerated artery was divided and ligated. Pulsatile bleeding came from the distal end. Because of the active retrograde bleeding the vein was left intact. The superficial femoral was then divided and ligated 1 centimeter distal to the profunda. When the casualty was evacuated 6 days after operation he had a foot drop, warm foot, and active plantar flexion.

No. 11755: On admission the leg was cold and pulseless. At operation 14 hours after injury a severed artery and vein were ligated. Soft part damage was severe. The superficial femoral artery was ligated as in the previous case. The extremity was disarticulated at the knee on the fifth postoperative day.

Tube anastomosis was done in 3 cases. No. 12052: On admission the leg was cold, pulseless, and motionless. At operation 13 hours after injury, soft part damage was moderate and the artery and vein were severed. A plastic tube was used to bridge a 5 centimeter gap in the artery and the vein was ligated. Because of tense enlargement of the leg incisions were made along the lateral aspects of the calf. Heparinization was started 12 hours after operation and discontinued after 15 hours because of excessive bleeding from the wounds. Due to misinterpretation of the tests for clotting time, the latter got beyond desirable limits. The postoperative course was stormy with mild generalized edema and extensive swelling of the affected extremity. A hematoma was evacuated from the wound. On the eighth day he had a pulmonary infarct and the common femoral vein was divided and ligated on the injured side. Over the entire period the leg remained warm, but the casualty was obviously toxic. On the seventeenth postoperative day a supracondylar amputation was done. The musculature of the leg was necrotic and the lumen of the artery about the tube had been obliterated by clot.

No. 12847: On admission the leg was cold and pulseless. At operation 24 hours after injury the artery was found severed, vein intact, and soft part damage moderate. Bleeding from the distal end of the vessel was scant. A glass tube was placed in the defect in the artery, and the vein was left intact. Seven days after operation an arteriogram was made; the dye went no further than the tube. The tube was removed on the sixth postoperative day and avascular portions of soleus and gastrocnemius were found and excised. Twelve days after operation the patient was evacuated with a warm, mobile foot.

No. 14765: Preoperatively the leg was warm and pulseless. At operation 17 hours after injury a lacerated artery and vein were found with moderate soft part damage. Bleeding from the distal segment was described as scant. The vein was divided and ligated. After division of the artery, a glass tube was inserted between the retracted ends. Because of edema, incisions were made along the lateral aspects of the calf on the third postoperative day. Necrotic soleus and extensor muscles were excised. Eight days posttrauma the foot was cold though evidence of gangrenous changes had not appeared. The outlook in this case was so poor that for statistical purposes it has been included among the amputations.

TABLE IV.—OPERATIVE PROCEDURES IN POPLITEAL ARTERY INJURIES

Operation	No.	Viable	Amputee
Ligation artery	2	1	1
Ligation artery and vein	14	3	11
Elective ligation	2	1	1
Tube anastomosis	3	1	2
Ligation vein	1	0	1
No operation	2	0	1

Ligation of the vein was done in patient No. 9530. A contused, thrombosed artery in a clinically avascular extremity was merely inspected and its companion vein ligated. Disarticulation followed.

No operation was done in 2 cases. Patient No. 1074 was admitted in irreversible shock.

No. 11848: A patient with a closed 4 day old, comminuted fracture of the distal end of the femur with posterior displacement was admitted with a gangrenous leg. Amputation was done through the fracture site after awaiting demarcation.

ANTERIOR AND POSTERIOR TIBIAL ARTERIES

We have included patients with concomittant injuries of these vessels in the series. There were 8, and 7 required amputation. In our experience isolated injuries of either artery were without incident. Associated fractures were present in 7 cases and soft part damage was severe in all cases. The condition of the extremity on admission was described in 5 cases as cold and pulseless. All were débrided, ligated, and immobilized in bivalved plaster encasements. Gas bacillus infection developed in 2 extremities and led to amputation in each instance. These patients received an average of 4 sympathetic blocks.

In the light of our experience, primary amputation may well have been the treatment of choice in some of these cases. It certainly would have been had we been unable to keep these men under observation.

AXILLARY ARTERY

There were 6 patients with severed axillary arteries in the group. The injury was localized to the third portion in 5 of the 6. In 1 the site was not specified. Amputation was necessary in 3 cases. Gas bacillus infection developed in 1 case. The process was confined to the biceps and was controlled by muscle excision. The extremity went on to extensive circulatory gangrene.

No. 10264: A severed artery was débrided and anastomosed with No. 0000 silk. The preoperative status of the extremity was not recorded. While it was warm at evacuation on the sixth day, a peripheral pulse was never palpable. Under the circumstances the result cannot be ascribed to the method.

BRACHIAL ARTERY

There were 40 brachial injuries with no deaths. In all but 2 cases the injury was distal to the profunda branch. In 5 cases amputation was done for circulatory gangrene. There were no gas bacillus infections. Thirty-seven per cent of the group had associated fractures whereas fractures were present in all the amputations. The time interval between injury and operation was 16 hours and was slightly less among those patients who came to amputation.

TABLE V.—OPERATIVE PROCEDURES IN BRACHIAL ARTERY INJURIES

	No.	Viable	Amputee
Ligation artery	17	15	2
Ligation artery and vein	17	16	1
Suture	3	3	
Anastomosis (suture)	1	1	
Débridement	1		1
Tube anastomosis	1		1

Ligation was carried out in 34 cases. Ligation of the companion vein or veins has long been considered a factor in diminishing the incidence of circulatory gangrene. The difference between ligation and nonligation is not appreciable in this series. Makins reports a series of brachial artery injuries treated by ligation. Of 17 cases in which the artery alone was ligated, 6 developed gangrene. In 7 cases artery and vein were ligated and only 1 became gangrenous. However, it must be pointed out that the two series are not strictly comparable in that Makins' includes late ligations for secondary hemorrhage, arterial aneurysms, and arteriovenous fistulas.

The 3 patients treated by suture were evacuated with mobile extremities. One case is particularly worth noting. This patient on admission had a cold and pulseless hand. Operation was done 12 hours after injury. A laceration of the brachial artery proximal to the profundus was found and sutured. There was an associated compound fracture of the humerus. Patient received 5 stellate blocks following surgery and relaxing incisions carried out in forearm due to the tenseness of the tissues. He was evacuated 4 days following surgery with a Volkmann's ischemic paralysis but with an apparently viable limb. No. 9158: The suture anastomosis is unfortunately lacking a preoperative description.

Tube anastomosis was done in 1 case. Patient No. 12850 had a shell fragment wound of the proximal third of the arm with extensive soft part damage, fracture, severed artery, vein, and nerves. The extremity was cold and pulseless. At operation 22 hours after injury the vein was ligated and a glass tube was inserted in the arterial defect. A weak radial pulse was present for 2 days. Then the circulation failed, gangrene appeared, and a supracondylar amputation was done on the fourth day. The blood supply to this stump was inadequate, infection developed, and reamputation through the fracture site was necessary. Collateral vessels were no doubt destroyed at the time of injury and, when the tube clotted, the blood supply ceased.

A brief summary of the 5 amputees is worth noting (Table V). No. 3545 was admitted with a wound to the elbow, resulting from a rifle bullet. The hand was cold and pulseless. There was also an associated fracture of the lower humerus and a penetrating wound of the chest. Ligation of the artery was done 12 hours after injury. Dry gangrene of the distal phalanges developed and amputation was carried out on the fifteenth postoperative day. A supracondylar amputation was done 10 days later because of circulatory gangrene.

No. 3553 was admitted with a machine gun wound of the elbow associated with fracture of the humerus. A tourniquet had been in place for 5 hours. Marked edema and mottling of the extremity were noted. The limb was cold and pulseless. The artery was found, severed, and was ligated. Patient was evacuated 1 week later with dry gangrene of the digits. It is assumed that amputation of the digits was done later.

No. 7153 was operated upon in another evacuation hospital and a ligation of the brachial artery and vein done. There was an associated fractured humerus and injuries to the median and ulnar nerves. Circulatory gangrene was evident on admission to our hospital 4 days later and an amputation through the fracture site was carried out.

No. 10283 had a bullet wound of midarm with a cold and pulseless extremity on admission. There was no evidence of bleeding. An associated fracture was present. Débridement was done and no surgery of the vessels was attempted. Patient was given the usual postoperative treatment with stellate blocks

Amputation through the lower arm was carried out several days later as a result of circulatory gangrene.

We do not feel that the initial procedure in this case was justified. The underlying pathology regarding the artery in this particular case was not noted. We believe that some type of arterial surgery should be done in every case of arterial injury.

No. 12850 suffered a shell fragment wound of the upper arm associated with extensive soft tissue damage which included injury to all three major nerves. There was an associated fracture. Hand was cold and pulseless on admission. A glass tube was inserted into the severed end of the artery and the vein was ligated (also severed). Supracondylar amputation was done 4 days later. Leucopenia developed on the sixth postoperative day as a result of sulfadiazine which was discontinued. On this eighth postoperative day, a second amputation was necessary through the fracture site as a result of circulatory gangrene in the stump with extensive infection. The glass tube was clotted when removed at the last procedure, but a weak radial pulse was present for 2 days following its insertion. No heparin was available for use at this time.

We thought it would be of interest to compare our results with another series of primary ligations as the majority of our cases were treated by primary ligation. From Makins' book on gunshot injuries of arteries written after the last war we have extracted a few figures. He has presented his statistics in terms of the number of cases of gangrene rather than as amputations. Where amputations were recorded they approximated the instances of gangrene. We have expressed the failures in our series as amputations.

TABLE VI.—COMPARISON OF RESULTS OF MAKINS AND AUTHORS

Artery	No.	Gangrene		No.	Amputations	
		No.	Per cent		No.	Per cent
Superficial femoral	43	11	25.6	21	13	62
Popliteal	28	8	28.5	16	12	75
Axillary	4	0		6	3	50
Brachial	5	3		34	3	

The figures reproduced from Makins' text are extracts from a larger series of ligations. Primary ligation during the First World War was apparently reserved for patients who were bleeding when admitted to a hospital installation. The majority of ligations were done at a later date for complications—secondary hemorrhage, arterial aneurysms, and arteriovenous fistulas.

CONCLUSIONS

1. A careful study of major peripheral vascular injuries is presented.

2. Our results of vascular injuries have been disappointing. Forty-two per cent developed circulatory gangrene which necessitated amputation before evacuation. We feel confident that amputation was carried out later on a few that apparently had viable limbs upon release from our hospital. Popliteal injuries especially carry a very poor prognosis.

3. We were not impressed with paravertebral sympathetic blocks in the treatment of these cases.

4. Severe associated soft tissue and bone damage increase the incidence of circulatory gangrene.

5. Saving of the patient's life should be the primary consideration in the plan of treatment. Long and tedious operative procedures are usually contraindicated in these cases as a result of the poor general condition of the patient.

6. The time interval between injury and operation is a major factor in the poor results obtained with intubation.

7. Gas gangrene was not an uncommon complication in vascular injuries and practically all resulted in amputation.

8. Skin temperatures and sensation are frequently misleading in judging viability of a limb. Active retrograde bleeding and motion of the toes or fingers are usually good prognostic signs, but not infallible. An elevated pulse rate and an anemia that showed no appreciable response to transfusion were almost constant findings in nonviable extremities.

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2. MAKINS, G. H. On Gunshot Injuries to the Blood Vessels. Baltimore: William Wood & Co., 1919.

EDITORIAL

SURGERY
Gynecology and Obstetrics

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NOVEMBER, 1946

MALARIA AND TRANSFUSIONS

SINCE the beginning there have been many hazards connected with the transfusion of blood. In the past, most of these have been considered in connection with the mechanics of the operation, the pyrogenic properties of the anticoagulant, the transmission of syphilis and the presence of isohemagglutinins. With the discovery of the Rh factor other hazards have been recognized, and more recently the possibilities of transmission of infectious hepatitis and malaria have received the attention of physicians.

The possibility of the inoculation of a patient accidentally with malaria as a result of blood transfusion has always been present in those countries and parts of countries in which malaria has been endemic. That the accident has happened, perhaps frequently, under such conditions, may be accepted without resort to the literature. On the other hand, a search of the literature will not reveal anywhere near the number of instances which must have occurred. This is natural; to many it would be like "washing one's linen in public" to publish such cases, and usually one finds only those reported in which the accident has occurred in the practice of another.

The deliberate transfer of malaria to persons suffering from general paresis is simply and readily accomplished by injecting small amounts of blood intramuscularly, subcutaneously or intravenously. It is interesting to note in passing that malaria produced by such methods, if the organism be Plasmodium vivax, is usually not severe and is readily controlled by moderate dosage of antimalarial drugs. Furthermore, relapses are rare. Even in approved hospitals, where such therapy is regularly carried out, the accidental transmission of malaria to some other patient, by the use of an unsterilized needle or syringe, has been observed, sometimes with most embarrassing results.

The more common accidental inoculation of plasmodia among addicts of drugs has been known for many years. These unfortunates, not knowing nor caring anything about sterile technique, frequently spread malaria among themselves by the use of common syringes, drug spoons and pipets. Even cities as far out of the normal malarial zone as New York annually record a number of cases of this bizarre phenomenon.

In spite of the fact that as a rule only a small percentage of cases is reported in which malaria is accidentally transmitted to the recipient of a transfusion, nevertheless one can find a number of recorded instances. Wright, after a review of the situation in 1938, reported this accident in thirty-eight instances. This group does not include fifty-four cases

reported by Wang and Lee in 1936, which occurred in 3,700 transfusions over a ten year period. These cases, together with another forty-five reported by Thoroughman, occurred in China, where malaria is endemic, but occasionally a few cases have been reported from regions where it is not endemic.

The recent report by Teasley focuses sharp attention on this hazard. The accident occurred in Minnesota during the winter and the patient was a man injured in a traffic accident who had not been outside the state. He had been previously transfused with blood from an ex-serviceman who had had several attacks of malaria contracted in the South Pacific area. Usually in such cases the symptoms begin in from eight to twenty-eight days, and the diagnosis is usually missed by the physician because he is not thinking of the possibility. Before the return of the ex-servicemen and women and the host of other personnel who found their way by one means or another into the area of the world invaded by war and in which malaria was endemic, malaria was known to most practitioners in northern states only as a textbook entity.

There are now a large number of persons, scattered throughout the United States, who harbor plasmodia in their blood and physicians fairly commonly see persons having their first attack after discontinuing suppressive treatment, or during one of their relapses. It is therefore obvious that some of these persons used as donors, may be capable of transmitting malaria, if he or she has been previously exposed to the bite of an infected anopheline mosquito.

Much study has been made of means of preventing the transmission of malaria by transfusion and it has been fairly well established that the parasite does not live long in drawn blood kept at body temperature, perhaps no longer than forty-eight hours. If blood is stored at between 4 and 6 degrees C., it may be considered safe after seven days. There are as yet no antiseptics known, which when added to the blood may be relied on to sterilize it and at the same time not be harmful to the blood itself.

A number of rules have been proposed to safeguard the patient being transfused. Some physicians believe that no person should be used as a donor if he has a history of malaria within fifteen years. Such a rule appears to be needlessly stringent and would preclude the possibility of finding enough donors to furnish the necessary blood for emergency use in some countries or sections of the country. On the other hand, there are those experienced malariologists who believe little or no attention should be paid to the donor with respect to his history of malaria. They point out that if the physician bears the matter in mind and notes signs and symptoms of malaria in his patient after transfusion, he should suspect malaria, examine the blood and, if it is positive, treat the patient accordingly. Such cases are easily controlled and there is rarely a relapse. To a physician more familiar with the possibilities of legal action than with malaria, such advice appears a bit naive. To such a one, the following rules will perhaps be more satisfactory: (1) If the prospective donor has been in a malarious area and has had an attack of malaria, he should not be used as a donor for at least two years after leaving the zone, provided that he discontinued suppressive treatment (atabrine or quinine) then and has not had any recurrences of malaria for at least two years prior to his donation. Before donation a thick smear should be examined and found to be negative. (2) If the prospective donor has been in a malarious area and has not had any attack of malaria and for the past year has not taken any suppressive treatment, he may be used provided a thick smear does not reveal

any parasites. The history of undiagnosed fever or a history which is questionable should provide the motive for the application of the first rule.

These rules will not absolutely prevent the occasional transmission of malaria by transfusion, but transfusions, like all operative procedures, carry some risks, and if the indications are for the administration of whole blood then this small risk must be assumed. The important thing is for the physician to be cognizant of the possibility of malaria following transfusions and to be prepared to diagnose and treat the disease. Under these circumstances, and with the application of the rules set forth, one may proceed with blood transfusions without any more real hazard than existed before the return of the men and women from other parts of the world.

T. B. MAGATH.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

AN outstanding contribution to dental and medical literature has been written by Dr. Burket.[1] The author, well qualified to compile this treatise, "discusses the many important relationships between oral and systemic disease" and encourages "a more universal and intimate co-operation between medical and dental practitioners," so that better health service is afforded the patient. Careful study of the material in this book will definitely impress upon the oral and dental surgeon that his obligation is more than the busy devotion to the mechanical aspect of his profession and will compel a renewal of his interests in the correlated subjects studied in his undergraduate years. The rewards will be great, for a wider horizon will appear, and his daily practice will be a constant stimulus rather than something that *can* become a distasteful chore.

To the medical man too there is offered food for thought. Too often his appreciation of the importance of the oral cavity is indifferent. Dr. Burket takes up this problem with understanding authority, and his outspoken pleas for a more lively effort in mutual appreciation and co-operation are timely and compelling.

The subject matter is well organized and very inclusive. Eleven sections are devoted to the different aspects of oral disease. Section 1 deals with medical-dental relations and the discussion of this important subject is complete. The value of histories and adequate examinations is stressed. Laboratory methods are included in sufficient detail to enlighten the earnest dental student and to help in the evaluation of reports.

Section 2 deals with the fusospirochetal infections which have become increasingly common and which are so often baffling in treatment. One of the chapters in this section deals with metallic poisoning and inflammatory reactions traced to dental materials.

No treatise on oral medicine would be complete without consideration of allergic manifestations, and this chapter is well done. That revealing and unruly organ, the tongue, is given first consideration in Chapter VIII of Section 3, an important and valuable chapter.

Dermatological lesions are always of acute interest to one attempting to make a diagnosis of oral lesions and forty pages are devoted to the dermatoses.

Section 4 discusses disease by systems. One hundred and seventy pages are devoted to a splendid résumé and evaluation of systemic disease. Sections dealing with nutritional deficiencies and diseases of metabolism, diseases of the blood and blood forming organs, syphilis, and the infectious granulomas follow.

Section 8 is devoted to the time honored subject, focal infection. It is an interesting, fair, and inclusive evaluation of the whole question. Of especial interest also are the discussions of oral pediatrics and gerodontics.

This entire volume represents a thorough and painstaking effort and is written with imagination. It is modern and designed to awaken both dentists and physicians to the need for more knowledge of a field too often neglected.

In the main, the illustrations are appropriate and satisfactory. Section 10 is devoted entirely to a colored atlas. The charts are graphic and valuable, and the regional diagnostic index is a distinct addition.

The bibliography is excellent and revealing to the casual reader.

Every oral surgeon will welcome the opportunity to consult and study this book, and undergraduates can use it profitably as a text.

FREDERICK W. MERRIFIELD.

IN 1930, the W. B. Saunders Company published a textbook of gynecology by Dr. Arthur H. Curtis. This first edition expressed the personal opinions of the author on most gynecological subjects. It was carefully planned and reflected the rich clinical experience of a master clinician and teacher. Since that time, in four later editions, the *Textbook of Gynecology*[2] has been kept up to date and has become more comprehensive. The most recent edition, as is true of those which preceded it, is divided into sections wherein the various subjects are completely covered. The text is agreeably readable and the numerous excellent photographs, photomicrographs and drawings (many of them by Tom Jones) add a great deal to the value of the book.

The first section, of which the coauthor is Barry J. Anson, deals with the anatomy of the female pelvis and perineum. In its preparation, the authors apparently employed painstaking care and accurate knowledge, similar to that which is so necessary for proper handling of surgical conditions in the region. The section, an excellent monograph, is illustrated by many new and original drawings.

Several sections deal with physiology relative to the genitalia in the various ages in a woman's life and

[1] ORAL MEDICINE; DIAGNOSIS, TREATMENT. By Lester W. Burket, D.D.S., M.D. With a section on ORAL ASPECTS OF AVIATION MEDICINE by Major Alvin Goldhush, D.D.S., M.S., A.U.S. Philadelphia, London, Montreal: J. B. Lippincott Co., 1946.

[2] A TEXTBOOK OF GYNECOLOGY. By Arthur Hale Curtis, M.D. 5th ed. Philadelphia and London: W. B. Saunders Co., 1946.

a sane appraisal of the value of hormones and of relevant knowledge of endocrinology is outlined.

The section on infections is complete and reflects the great interest and original work of the author in this field. In the chapters on chemotherapy, fever therapy and surgery are presented the latest advances in treatment of the various types of infections.

Sections dealing with benign and malignant tumors which are found in the various genital organs, as well as sections which have to do with all pathologic conditions that result from childbirth, are complete and indications for surgical treatment are clearly explained. Sterility, leucorrhea, pruritus vulvae, vaginismus, and so on are thoroughly considered and rational treatment is outlined.

The extensive and up-to-date bibliography which follows each chapter constitutes evidence of the months of careful planning and fact gathering that were expended on the work.

This excellent book of 718 pages is clear and thorough. It is recommended not only for the student and interne but for the general surgeon and the gynecologist. J. C. MASSON.

DEDICATED to the memory of George Brown, whose untimely death in 1935 has been deeply mourned by those who knew his pioneering activities and were ever exposed to his dynamic personality, *Peripheral Vascular Diseases*[1] is a monumental piece of work. The authors, Dr. Brown's former associates, have written a large volume, unquestionably the most extensive and the most up-to-date presentation of the subject. Although equipped with an excellent bibliography, the monograph essentially represents the practice of the Mayo Clinic, with a critical appraisal of methods practiced elsewhere. In 31 chapters profusely illustrated the authors have fully covered the field. They have purposely omitted the discussion of hypertension, a subject which truly needs separate treatment: the technique of sympathectomy, also, is not discussed, although other surgical procedures such as vein ligation, amputation, embolectomy, the treatment of arteriovenous fistulas, skin grafting, and the surgical treatment of lymphedema are fully described. Since frequent reference is made in the text to the indications for and value of sympathectomy in peripheral vascular disease it is to be hoped that in future editions the authors may reconsider this purposeful omission.

Some of the chapters are truly gems of excellent organization, lucid style, and clear presentation. Such are the discussions on capillary microscopy, on the sweating mechanism, on arteriosclerosis, on thromboangiitis obliterans. Others, while containing valuable data and obviously reflecting vast personal experience, are loosely organized, repetitious, and could be profitably shortened. All in all, a happy medium has been found between a didactic textbook for students and a postgraduate course of instruction for the medical practitioner. For the surgeon interested in peripheral vascular disease this is material which he cannot afford to miss. GEZA DE TAKATS.

[1]PERIPHERAL VASCULAR DISEASES. By Edgar V. Allen, B.S., M.A., M.D., M.S., Nelson W. Barker, B.A., M.D., M.S., and Edgar A. Hines, Jr., M.D., B.S., M.A., M.S., with associates in the Mayo Clinic and Mayo Foundation. Philadelphia, London: W. B. Saunders Co., 1946.

BREAKING a long interval in which there have been no weighty additions to the list of textbooks of embryology written in the English language, first an Anglo-American contribution (Hamilton, Boyd and Mossman) and then an American volume[2] appear in quick succession. Professor Patten's book has been in preparation for more than a decade, and its appearance has been awaited with pleasurable anticipation since his skillful handling of simpler college texts on the development of the chick and pig left no doubt that a more pretentious volume on man would bring a fresh treatment to an old subject and a mature management of material that is inherently difficult to present clearly.

It is a commonplace, but worth re-emphasizing, that embryology deals with processes occurring both in time and in space. It is the sort of thing for which a stereoscopic motion picture provides the only theoretically perfect medium of presentation; the difficulties faced by the textbook writer can be appreciated by anyone who will attempt to write simple and clear directions for tying a bow knot. Professor Patten's long pedagogical experience has guided him into ways of presentation which find an outlet in this text. His gift of clear, forceful expression and a showmanlike handling of illustrations are at their best in this well balanced product.

There are certain things that this book does not attempt to cover, such as genetic considerations, comparative embryology, experimental embryology and various aspects of general embryology. Laboratory aid is omitted, except that some stages of pig embryos are included for comparison in a chapter on "Early Differentiation of the Body and Establishing of Organ Systems." These omissions liberate space and the 776 pages allotted can thereby contain rather full details and illustrations of the subjects treated. Actually the extra-full page as set up gains additional space equivalent to a book of 832 pages by ordinary standards. Judicious use has been made of such an uncommon amount of space assigned to a minor course in the medical curriculum.

The text is divided into 19 chapters which merge without larger headings. Any plan by which the 11 chapters dealingwith organogenesis take sequence is not apparent, but order of a traditional kind is perhaps not one of great importance. Anomalies are treated in a plenary manner in comparison with the limited space accorded them in most general texts. There is a selected bibliography inserted as an appendix. Its entries are grouped under topics corresponding to the chapters, and its content is restricted to items dealing with human development. This type of bibliography gives standard sources of reference but fails to indicate which ones deal with fore-

[2]NEW HUMAN EMBRYOLOGY. By Bradley M. Patten. Philadelphia: The Blakiston Co., 1946.

front advances and the still unsettled regions of the subject. There is an adequate index.

There can be no question that Professor Patten has presented a work of merit both in description and in generous pictorial embellishment. The text is a reliable exposition of the morphogenetic features of development. The illustrations accomplish their purpose, and often do so with notable success; especially is this true in some of the sequential sets. Color is used in 53 of the 446 figures. The clinician and advanced medical student will find this book to be a helpful source of information since it occupies a position intermediate between the more elementary and the more encyclopedic works. It will be interesting to see how the routine medical courses with but 50 to 75 hours available for didactic and laboratory work will handle a volume of this size devoted to the purely human phases of development. The preface makes clear that the text is not intended for the general college course of embryology. L. B. Arey.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Early Ambulation and Related Procedures in Surgical Management. By Daniel J. Leithauser, M.D., F.A.C.S. Springfield: Charles C Thomas, 1946.

Penicillin its Practical Application. By Sir Alexander Fleming, M.B., B.S., F.R.C.P., F.R.C.S., F.R.S. Philadelphia: The Blakiston Co., 1946.

The Chest; a Handbook of Roentgen Diagnosis. By Leo C. Rigler, M.D. Chicago: The Year Book Publishers, Inc., 1946.

Treatment of Bronchial Asthma. By Vincent J. Derbes, M.D., and Hugo T. Engelhardt, M.D., F.A.C.P. Philadelphia, London, Montreal: J. B. Lippincott Co., 1946.

Tumores Broncogenicos. By Hernan D. Aguilar. Buenos Aires: Libreria y Editorial, "El Ateneo," 1946.

The Technical Minutiae of Extended Myomectomy and Ovarian Cystectomy. By Victor Bonney, M.S., M.D., B.Sc. (Lond.), F.R.C.S. (Eng.), Hon. F.R.A.C.S., M.R.C.P. (Lond.). New York, London: Paul B. Hoeber, Inc., 1946.

Clinica y Laboratorio. By Dr. Gustavo Pittaluga, Dr. Enrique Galan, and Dr. Antonio Guernica. Havana, Cuba: M. V. Fresneda 1946.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

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PRELIMINARY PROGRAM FOR THE 1946 CLINICAL CONGRESS IN CLEVELAND—DECEMBER 16 TO 20

THE five-day Clinical Congress of the American College of Surgeons will open in Cleveland on Monday morning, December 16, at 9:30 o'clock. Headquarters will be in the Cleveland Public Auditorium, and the Statler and Cleveland hotels, with most of the sessions, except the clinics at the hospitals, being held in the Auditorium. The programs, outlines of which appear on following pages, are practically complete. They show an interesting and varied plan of presenting wartime and postwar developments in surgery through the media of operative and non-operative clinics, demonstrations, symposia, panel discussions, forums, motion pictures, and exhibits.

The program of the Congress will include the twenty-fifth annual Hospital Standardization Conference, which will convene during the first four days, with morning and afternoon sessions on Monday through Thursday beginning at 9:00 o'clock and 2:00 o'clock, respectively, and evening meetings on Tuesday and Wednesday beginning at 7:30.

The first event on the Clinical Congress program will be the General Assembly for surgeons and hospital representatives which will be held on Monday morning. The Monday afternoon program will include two panel discussions, one beginning at 1:30, the other at 3:00; clinics in the local hospitals; a hospital conference; and a surgical film exhibition. The Monday evening program will be devoted to the Presidential Meeting.

On the four mornings beginning Tuesday at 8:00 o'clock, operative and non-operative clinics will be held in the local hospitals. From 9:00 to 12:30 on each of these mornings, the Forum on Fundamental Surgical Problems will meet in the Music Hall of the Cleveland Public Auditorium. From 9:30 to 12:30, Tuesday through Thursday, hospital conferences will be held in the Ballroom of the Auditorium. At 10:00 o'clock on Tuesday through Friday, surgical film exhibitions are scheduled. At 11:00 o'clock on Tuesday through Thursday, panel discussions on ophthalmology and on otolaryngology will be held.

Afternoon programs each day include clinics; two major panel discussions from 1:30 to 3:00 and from 3:30 to 5:00 o'clock respectively (except Wednesday when only the 3:30 panel will be held); hospital conferences (Monday through Thursday); and surgical film exhibitions. In addition, on Tuesday afternoon at 2:00 o'clock there will be a symposium on fractures and other traumas; on Wednesday afternoon at 2:00 o'clock a symposium on cancer; on Thursday afternoon at 3:00 o'clock a symposium on graduate training in surgery; and on Friday afternoon six concurrent specialty panels from 2:00 to 4:00 o'clock in the fields of obstetrics, plastic surgery, neurological surgery, thoracic surgery, urology, and orthopedic surgery.

Symposia on general surgery will be held Tuesday, Wednesday, and Thursday evenings; symposia on ophthalmology and on otorhinolaryngology on Tuesday and Thursday evenings; and a symposium on plastic surgery of the head and neck on Wednesday evening. The concluding event will be the Convocation to be held on Friday

evening at 8:15 o'clock in the Music Hall of the Cleveland Public Auditorium.

CLINICAL PROGRAM

With the aid of a representative in each hospital, a committee on arrangements appointed by the surgeons of Cleveland, with Dr. Thomas E. Jones as chairman and Dr. John W. Holloway as secretary, has prepared a preliminary program which is published in succeeding pages. This consists of operative and non-operative clinics, demonstrations, presentation of cases, symposia, pathological conferences, and surgical staff conferences. Seventeen hospitals are listed in this preliminary outline, and the program for each hospital is classified according to specialties. A wide range of subjects is covered, including general surgery, pathology, fractures and other traumas, thoracic surgery, tumor surgery, plastic surgery, ophthalmology, otolaryngology, neurosurgery, vascular surgery, orthopedic surgery, obstetrics and gynecology, genitourinary surgery, oral surgery, and roentgenology. The clinics and demonstrations will be held in the hospitals every morning and afternoon beginning Tuesday, December 17.

A complete detailed clinical program for each day will be posted in the form of bulletins at headquarters in the Cleveland Public Auditorium during the afternoon of the preceding day and distributed in printed form each morning.

HOSPITALS AND REPRESENTATIVES

City Hospital	J. H. Lazzari
Cleveland Clinic	Robert S. Dinsmore
Evangelical Deaconess	Oliver A. Weber
Fairview Park Hospital	W. E. Smith
Glenville Hospital	Jacob E. Tuckerman
Grace Hospital	L. J. Sternicki
Huron Road Hospital	Henry W. Brown
Lakeside Hospital	Frederick R. Mautz
Lakewood Hospital	C. Lee Graber
Lutheran Hospital	Frank S. Gibson
MacDonald House	R. L. Faulkner
Mt. Sinai Hospital	Rudolph S. Reich
Polyclinic Hospital	Henry A. Schlink
St. Alexis Hospital	John F. Corrigan
St. Ann's Hospital	E. P. Monaghan
St. John's Hospital	Farrell T. Gallagher
St. Luke's Hospital	Donald M. Glover
St. Vincent Charity	Oliver A. Weber
U. S. Marine Hospital	Mark E. Meyers
U. S. Veterans Hospital	Dr. Partington
Woman's Hospital	Ernest Cox

GENERAL ASSEMBLY

The Clinical Congress will open with a general assembly in the Music Hall of the Cleveland Public Auditorium at 9:30 o'clock on Monday morning. Dr. W. Edward Gallie, President of the College, will preside at this meeting which will be the opening session of the twenty-fifth annual Hospital Standardization Conference. Dr. Irvin Abell, Chairman of the Board of Regents, will report on the progress of the 1946 Hospital Standardization survey, final results of which, including the Approved Lists of hospitals, cancer clinics, and approved hospitals for graduate training in surgery, will be announced at the end of the year. Major General Charles R. Reynolds (Ret.), Director of the Department of Graduate Training in Surgery, will discuss the "Hospital Standardization Program as Foundation for Graduate Training in Surgery."

An address on "Maintaining our Voluntary Hospital System" will be given by Rev. Monsignor Maurice F. Griffin of Cleveland. Dr. Evarts A. Graham of St. Louis, Professor of Surgery, Washington University School of Medicine and a member of the Commission on Hospital Care, will present "Pertinent Findings from the National Survey by the Commission on Hospital Care."

The program will conclude with a discussion of the nursing problem. Dr. Howard C. Naffziger of San Francisco, Professor of Surgery, University of California Medical School and Chairman of the Committee on Nursing, American Surgical Association, will present the viewpoint of the surgeon. Dr. Robin C. Buerki of Philadelphia, Director, University Hospitals and Dean of the Graduate School of Medicine, University of Pennsylvania, will present the viewpoint of the hospital administrator. Lucile Petry of Washington, Director of Nursing, United States Public Health Service, will present the viewpoint of the nurse.

PRESIDENTIAL MEETING

The Presidential Meeting will be held on Monday evening in the Music Hall of the Auditorium. The officers, regents, and honorary guests will participate in the processional. Dr. W. Edward Gallie, President of the College, will preside. The address of welcome will be given by Dr. Thomas E. Jones, Chairman of the Cleveland Executive Committee; foreign and other guests will be introduced by Dr. Arthur W. Allen, Vice Chairman, Board of Regents; and Doctor Gallie will deliver the Presidential Address. An inaugural ceremony will be held for the incoming officers: President, Dr. Irvin Abell of Louisville; First Vice President, Dr. Leland S. McKittrick of Boston; Second Vice-President, Dr. F. Phinizy Calhoun of Atlanta.

The first Martin Memorial Lecture will be a feature of the Presidential Meeting this year. Dr. Edward D. Churchill of Boston has accepted the

invitation to give this lecture. The lectureship was established upon motion of the Board of Regents at its midyear meeting on April 1, as a memorial to both Dr. Franklin H. Martin and Mrs. Martin. The founder of the College and of SURGERY, GYNECOLOGY AND OBSTETRICS, was joined by his wife in making the College the beneficiary at their deaths of the Journal, together with its physical plant operated by the Surgical Publishing Company of Chicago, of which they were the owners. Doctor Martin died in 1935 and Mrs. Martin in 1945.

The Martin Memorial Lecture, to be given annually during the Clinical Congress, and dealing with a scientific subject of the author's choosing, supplants the Annual Oration in Surgery.

CONVOCATION

The Convocation will be held on Friday evening. It will open with a processional of officers, regents, governors, and initiates. The President, Dr. Irvin Abell, will preside, confer the honorary fellowships, and present the candidates for fellowship.

An Assembly of Initiates will be held at 10:30 o'clock on Friday morning in order to instruct them in the Convocation procedure and other matters.

Several hundred surgeons who have been received into fellowship *in absentia* during the war years in which no Convocation was held, will be added to the number of initiates for the current year who will be present to participate in the initiation ceremonies. The Convocation and in fact the meetings throughout the Congress will be occasions for reunion with many Fellows who have recently returned from service with the military forces.

FORUMS ON FUNDAMENTAL SURGICAL PROBLEMS

The Forums on Fundamental Surgical Problems will be conducted on Tuesday, Wednesday, Thursday, and Friday mornings from 9:00 to 12:30 o'clock. Included in them will be brief reports of original clinical and experimental observations relating to the broad aspects of surgery and the surgical specialties. No prepared discussions of the reports are planned, but questions and comments will be invited. Especially keen interest is expected in these sessions this year because of the accumulation of the results of 5 years of work since the last Clinical Congress. Dr. Owen H. Wangensteen of Minneapolis, chairman of the committee which is planning the program, is working toward representation of as many as possible of the various university departments of surgery in this presentation of clinical and experimental research work.

The enlistment of the interest of young men who are doing original work, through the forums, is one of the most beneficial results of these sessions which are now considered to be an indispensable feature of every Clinical Congress.

GENERAL SURGERY

The evening symposia in general surgery on Tuesday, Wednesday, and Thursday, will be held at 8:00 o'clock in the Music Hall of the Public Auditorium. The subject for Tuesday night is "Care of the Patient Before and After Operation." The subject for Wednesday night is "Venous Thrombosis and Prevention of Pulmonary Embolism," to be followed by the annual Fracture Oration which will be delivered by Dr. Edwin W. Ryerson of Chicago, the subject being "Modern Methods in the Treatment of Fractures." The subject for Thursday night is "Antibiotic and Chemotherapeutic Agents in Surgery."

The subjects for the afternoon panel discussions in general surgery on Monday through Friday afternoons are as follows: "Rehabilitation of the Surgical Patient and Early Ambulation" on Monday at 1:30; "The Treatment of Cancer of the Large Bowel" on Monday at 3:30; "Thiouracil in Thyroid Disease" on Tuesday at 1:30; "Recent Trends in the Management of Carcinoma of the Cervix" on Tuesday at 3:30; "Anesthesia" on Wednesday at 1:30; "Protein Metabolism in the Surgical Patient" on Wednesday at 3:30; "Spinal Cord Injuries" on Thursday at 3:30; "Surgery of the Stomach" on Friday at 1:30; and "Surgery of the Vascular System" on Friday at 3:30.

OPHTHALMOLOGY

The evening symposia in ophthalmology on Tuesday and Thursday will be held at 8:00 o'clock in South Hall A of the Cleveland Public Auditorium. "Orbital Reconstruction Including Prosthesis" will be the subject on Tuesday evening and "Recent Advances in Ophthalmology" will be the subject on Thursday.

Panel discussions on "Retinal Detachment," "Glaucoma," and "Keratoplasty," respectively, are scheduled for Tuesday, Wednesday, and Thursday mornings at 11:00 o'clock.

OTORHINOLARYNGOLOGY

The evening symposia in otorhinolaryngology on Tuesday and Thursday will be held at 8:00 o'clock in South Hall B of the Cleveland Public Auditorium. "Treatment of Deafness" will be the

subject on Tuesday evening and "Surgery of the Nasal Accessory Sinuses" on Thursday.

Panel discussions on "The Treatment of Meniere's Disease," "Osteomyelitis of the Skull," and "Rehabilitation of the War Deafened," respectively, are scheduled for Tuesday, Wednesday, and Thursday mornings at 11:00 o'clock.

PLASTIC SURGERY OF THE HEAD AND NECK

A special symposium on plastic surgery of the head and neck will be held on Wednesday evening at 8:00 o'clock in South Hall A. This symposium will be of interest to many general surgeons as well as to ophthalmologists and otorhinolaryngologists. The following subjects will be discussed: "Cancellous Bone Grafts to the Jaw;" "Repair of Nasal Defects with Free Composite Grafts of Skin and Cartilage from the Ear;" "Studies in the Anatomy and the Repair of Cleft Palate;" and "Preservation of Function Following Resections of Jaw Tumors."

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

"Advances from War Experience" is the general theme of the Symposium on fractures and other traumas which has been arranged by Dr. Robert H. Kennedy of New York, chairman of the Committee on Fractures and Other Traumas of the American College of Surgeons. This meeting will be held at 2:00 p.m. on Tuesday in the Ballroom of the Public Auditorium. From the outline shown on a succeeding page it will be seen that an impressive panel of 19 speakers will discuss different phases of the subject and that their presentations will be summarized by Vice Admiral Ross T. McIntire, Surgeon General of the United States Navy; Major General Norman T. Kirk, Surgeon General of the United States Army; and Major General Paul R. Hawley, Chief Medical Director of the United States Veterans Administration.

SYMPOSIUM ON CANCER

A symposium on cancer, sponsored by the Cancer Committee of the College, will be held at 2:00 o'clock on Wednesday afternoon in the Ballroom of the Cleveland Public Auditorium. Besides the talks on surgical problems in this field, there will be discussion of organization for cancer control and reports on further 5 year cures of cancer.

GRADUATE TRAINING IN SURGERY

A panel discussion on graduate training in surgery and the surgical specialties will be held Thursday afternoon at 3:00 o'clock in Club Room B. Discussion will be from the standpoints of the American College of Surgeons, the basic medical sciences, the Veterans Administration, the university-connected hospital, and the hospital which is not connected with a university.

ANNUAL MEETING OF FELLOWS

The annual meeting of the Governors and Fellows will be held on Thursday afternoon at 1:45. There will be election of officers and governors. The annual meeting affords the Fellows of the College an opportunity to hear reports of officials on the work of the organization, and to learn how it has not only raised the professional and ethical standards of surgery, but has also promoted good hospitalization and general improvement in the practice of medicine in the United States and Canada. Each Fellow has a personal part in this work and may extend the influence of the College materially in his local community. Hospital Standardization alone offers him unlimited opportunity to provide better medical care for his patients in the hospital in which he works, through continuous progress in applying the principles of the Minimum Standard which insure the best care of the patient.

Every Fellow will want to attend this important meeting, at which reports will be presented on finances; Hospital Standardization; Graduate Training in Surgery; Medical Motion Pictures; Publications; Public Relations; Library and Literary Research; the work of the state and provincial credentials committees, committees on applicants, and the Committee on History Reviews; Sectional Meetings, 1946; and the Department of Clinical Research, including cancer clinics, Medical Service in Industry, the Committee on Cancer, and the Committee on Fractures and other Traumas. Dr. Abell will report on administration of the College, staff changes, and retirements, and Dr. Arthur W. Allen, Vice-Chairman of the Board of Regents will discuss "Fellowships, Obligations and Opportunities."

MEDICAL MOTION PICTURES

The latest available films showing surgical procedures and related subjects will be shown in the medical motion picture exhibits which will be held daily in the Little Theatre of the Cleveland Public Auditorium. These are a much appreciated feature of the Clinical Congress. Despite the decrease in production of such films during the war period, a surprising number of new pictures on varied subjects are being received by the American College of Surgeons for review. Both sound

and silent, colored films will be shown, all of which have been approved by the Committee on Medical Motion Pictures.

TECHNICAL AND SCIENTIFIC EXHIBITION

The technical exhibit, together with the registration and clinic ticket bureaus, will be held in the Arena of Cleveland Public Auditorium. Leading manufacturers of surgical instruments, x-ray apparatus, sterilizers, operating room lights, ligatures, dressings, hospital apparatus and supplies of all kinds, pharmaceuticals, and publishers of medical books will be represented in the exhibition. The technical exhibits will demonstrate many of the newer features learned from our experience in the war.

ADVANCE REGISTRATION

The hospitals of Cleveland afford accommodations for a considerable number of visiting surgeons. However, in order to insure against overcrowding, attendance at the Congress will be limited to the number that can be comfortably accommodated at the meetings and also by accommodations in the hotels. It is therefore expected that surgeons who wish to attend the Congress will register in advance.

Fellows of the College whose dues are paid to December 31, 1945, initiates of the classes of 1942, through 1946, and Fellows in military service will not be required to pay a registration fee for the Clinical Congress in Cleveland. Checks that have already been sent for the New York Congress are being returned. For endorsed junior candidates the fee is $5.00. Surgeons, not Fellows, who attend as invited guests of the College, will pay a registration fee of $10.00.

CLEVELAND HOTELS AND THEIR RATES

Although the hotel situation in Cleveland seems more favorable than in many other cities, it is nevertheless essential to make reservations as far in advance as possible. Following is a list of the principal hotels:

	No. of Rooms	Rates
The Alcazar, Surrey and Derbyshire Roads	200	$3.00 up
Hotel Allerton, E. 13th St. & Chester Ave.	600	1.75 "
Hotel Auditorium, E. 6th & St. Clair Ave.	300	2.00 "
Belmont Hotel, Euclid Ave. at E. 40th St.	200	2.00 "
Hotel Bolton Square, Carnegie at E. 89th St.	300	2.50 "
Carter Hotel, 1012 Prospect Ave.	600	3.00 "
Hotel Cleveland, Public Sq. & Superior Ave.	1,000	3.00 "
Fenway Hall Hotel, Euclid Ave. at Univ. Circle	400	3.50 "
The Hollenden, Superior Ave. at E. 6th St.	1,000	3.00 "
Lake Shore Hotel, 12506 Edgewater Dr.	450	3.50 "
New Amsterdam Hotel, Euclid Ave. at E. 22d St.	300	2.00 "
Hotel Olmsted, E. 9th St. & Superior Ave.	250	2.00 "
Park Lane Villa, E. 105th & Park Lane Ave.	400	3.00 "
Sovereign Hotel, 1575 E. Blvd.	300	2.50 "
Hotel Statler, Euclid Ave. at E. 12th St.	1,000	3.00 "
Sterling Hotel, Prospect Ave. at E. 30th St.	200	2.25 "
Tudor Arms Hotel, Carnegie at E. 107th St.	200	3.00 "
Wade Park Manor, E. 107th & Park Lane	400	3.00 "
The Westlake, Blount St., Rocky River	400	2.50 "

CLINICAL CONGRESS PROGRAM IN BRIEF

UNLESS OTHERWISE DESIGNATED, THE MEETING ROOMS LISTED ARE IN THE CLEVELAND PUBLIC AUDITORIUM

Monday

9:30 General Assembly for Surgeons and Hospital Representatives, Music Hall
1:30 Panel Discussion, Music Hall
2:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
2:00 Hospital Conference, Ballroom
1:00 Surgical Film Exhibition (General), Little Theatre
3:30–5:00 Panel Discussion, Music Hall
8:15 Presidential Meeting, Music Hall

Tuesday

8:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
9:00 Forum on Fundamental Surgical Problems, Music Hall
9:30 Hospital Conference, Ballroom
9:30 Surgical Film Exhibition (Ophthalmology and Otorhinolaryngology), Little Theatre
10:00 Surgical Film Exhibition (General), Little Theatre
11:00 Panel Discussions
Ophthalmology, South Hall A
Otorhinolaryngology, South Hall B
1:30 Panel Discussion, Music Hall
2:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
2:00 Hospital Conference, Club Room B
2:00 Symposium on Fractures and Other Traumas, Ballroom
2:00 Surgical Film Exhibition (General), Little Theatre
3:30 Panel Discussion, Music Hall
7:00 Surgical Film Exhibition (Ophthalmology and Otorhinolaryngology), Little Theatre
7:30 Hospital Conference, Ballroom, Hotel Statler
8:00 Symposium, General Surgery, Music Hall
8:00 Symposium, Ophthalmology, South Hall A
8:00 Symposium, Otorhinolaryngology, South Hall B

Wednesday

8:00 Meeting of Cancer Committee, Tavern Room, Hotel Statler
8:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
9:00 Forum on Fundamental Surgical Problems, Music Hall
9:30 Hospital Conference, Ballroom
9:30 Surgical Film Exhibition (Ophthalmology and Otorhinolaryngology), Little Theatre
10:00 Surgical Film Exhibition (General), Little Theatre
State and Provincial Committees:
9:30 Judiciary Committees, Ballroom, Hotel Statler
10:00 Executive Committees, Ballroom, Hotel Statler
10:30 Credentials Committees and Committees on Applicants, Ballroom, Hotel Statler
11:00 Panel Discussions
Ophthalmology, South Hall A
Otorhinolaryngology, South Hall B
12:00 Meeting of Board of Governors, Lattice Room, Hotel Statler
1:30–3:00 Panel Discussion, Music Hall
2:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
2:00 Symposium on Cancer, Ballroom
2:00 Surgical Film Exhibition (General), Little Theatre
2:30 Hospital Conference, Club Room B
3:30–5:00 Panel Discussion, Music Hall
7:00 Surgical Film Exhibition (Ophthalmology and Otorhinolaryngology), Little Theatre
7:30 Hospital Conference, Ballroom, Hotel Statler
8:00 Symposium, General Surgery, Music Hall
8:00 Symposium, Plastic Surgery of the Head and Neck, South Hall A

Thursday

8:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
9:00 Forum on Fundamental Surgical Problems, Music Hall
9:30 Hospital Conference, Ballroom
9:30 Surgical Film Exhibition (Ophthalmology and Otorhinolaryngology), Little Theatre
10:00 Surgical Film Exhibition (General), Little Theatre
11:00 Panel Discussions
Ophthalmology, South Hall A
Otorhinolaryngology, South Hall B
1:30 Adjourned Meeting, Governors, Music Hall
1:45 Annual Meeting, Fellows, Music Hall
2:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
2:00 Hospital Conference, Ballroom
3:00 Panel Discussion—Graduate Training in Surgery, Club Room B
3:30 Panel Discussion, Music Hall
3:30 Surgical Film Exhibition (General), Little Theatre
3:30 National and Regional Fracture Committees, Club Room C
4:00 Committee on the Library, Tavern Room, Hotel Statler
7:00 Surgical Film Exhibition (Ophthalmology and Otorhinolaryngology), Little Theatre
8:00 Symposium, General Surgery, Music Hall
8:00 Symposium, Ophthalmology, South Hall A
8:00 Symposium, Otorhinolaryngology, South Hall B

Friday

8:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
9:00 Forum on Fundamental Surgical Problems, Music Hall
9:30 Surgical Film Exhibition (Ophthalmology and Otorhinolaryngology), Little Theatre
10:00 Surgical Film Exhibition (General), Little Theatre
10:30 Assembly of Initiates, Ballroom
1:30 Panel Discussion, Surgery of the Stomach, Music Hall
2:00 Clinics, Demonstrations and Group Conferences, Selected local hospitals
2:00 Panel Discussions
Obstetrics, South Hall A
Plastic Surgery, Club Room C
Neurological Surgery, Club Room A
Thoracic Surgery, South Hall B
Urology, South Hall C
Orthopedic Surgery, Club Room B
2:00 Surgical Film Exhibition (General), Little Theatre
3:30 Panel Discussion, Surgery of the Vascular System, Music Hall
8:15 Convocation, Music Hall

GENERAL ASSEMBLY

Monday, 9:30 a.m.—Music Hall, Public Auditorium

Opening Session of Clinical Congress—General Assembly
W. EDWARD GALLIE, M.D., Toronto; President, American College of Surgeons, Presiding.
Preliminary Statement. W. EDWARD GALLIE, M.D., Toronto.
Presentation of Report of Field Activities of the American College of Surgeons for 1946, and Summary of 28 Years of Hospital Standardization.
IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents, American College of Surgeons.
Maintaining our Voluntary Hospital System. Right Reverend Monsignor MAURICE F. GRIFFIN, Cleveland.
Pertinent Findings from the National Survey by the Commission on Hospital Care.
EVARTS A. GRAHAM, M.D., St. Louis; Professor of Surgery, Washington University School of Medicine; Member of Commission on Hospital Care.
The Nursing Problem:
The Viewpoint of the Surgeon.
HOWARD C. NAFFZIGER, M.D., San Francisco; Professor of Surgery, University of California Medical School; Chairman, Committee on Nursing, American Surgical Association.
The Viewpoint of the Administrator.
ROBIN C. BUERKI, M.D., Philadelphia; Director, University Hospitals and Dean of the Graduate School of Medicine, University of Pennsylvania.
The Viewpoint of the Nurse.
LUCILE PETRY, R.N., Washington; Director of Nursing, United States Public Health Service.

PRESIDENTIAL MEETING

Monday, 8:15 p.m.—Music Hall, Public Auditorium

W. EDWARD GALLIE, M.D., Toronto; President, American College of Surgeons, Presiding.
Processional—Officers, Regents and Honorary Guests.
Invocation.
BEVERLEY D. TUCKER, D.D., LL.D., Cleveland; Bishop of the Diocese of Ohio.
Address of Welcome.
THOMAS E. JONES, M.D., Cleveland; Chairman, Committee on Arrangements.
Introduction of Foreign and other Guests.
ARTHUR W. ALLEN, M.D., Boston; Vice Chairman, Board of Regents.
Address of the Retiring President. Ideals in Surgery.
W. EDWARD GALLIE, M.D., Toronto.
Inauguration of Officers:
President: IRVIN ABELL, M.D., Louisville.
First Vice President: LELAND S. MCKITTRICK, M.D., Boston.
Second Vice President: F. PHINIZY CALHOUN, M.D., Atlanta.
The Martin Memorial Lecture. The American Surgeon, Army of the United States.
EDWARD D. CHURCHILL, M.D., Boston. John Homans Professor of Surgery, Harvard University. Recessional.

CONVOCATION

Friday, 8:15 p.m.—Music Hall, Public Auditorium

IRVIN ABELL, M.D., Louisville; President, Presiding.
Processional. Officers, Regents and Initiates.
Invocation.
Right Reverend Monsignor MAURICE F. GRIFFIN, Cleveland.
Conferring of Fellowships by the President.
IRVIN ABELL, M.D., Louisville.
Fellowship Pledge. Recital by the Initiates.
Conferring of Honorary Fellowships. The President.
Fellowship Address. The Education of a Physician.
REUBEN G. GUSTAVSON, Ph.D., Omaha; Chancellor, University of Nebraska
Recessional.
Reception by the Officers and Regents for the Initiates and Fellows.

EVENING SESSIONS

GENERAL SURGERY

Symposia

Tuesday, 8:00 p.m.—Music Hall, Public Auditorium

IRVIN ABELL, M.D., Louisville; President, American College of Surgeons, Presiding

Care of the Patient Before and After Operation

Fluid and Electrolyte Balance. CARL MOYER, M.D., Eloise, Michigan

Nutritional Preparation of the Substandard Risk Patient. RICHARD L. VARCO, M.D., Minneapolis

Use of Blood and Blood Substitutes. JOHN D. STEWART, M.D., Buffalo

Blood Transfusion Problems, with Special Reference to the Rh Factor. ALEXANDER S. WIENER, M.D., Brooklyn

Safe Conduct of the Patient Through Operation. OWEN H. WANGENSTEEN, M.D., Minneapolis

Wednesday, 8:00 p.m.—Music Hall, Public Auditorium

PAUL B. MAGNUSON, M.D., Chicago; Secretary, American College of Surgeons, Presiding

Venous Thrombosis and Prevention of Pulmonary Embolism

Interruption of Deep Veins. ARTHUR W. ALLEN, M.D., Boston

Sympathetic Nerve Block. ALTON OCHSNER, M.D., New Orleans

Anticoagulants. D. W. GORDON MURRAY, M.D., Toronto.

Fracture Oration

Modern Methods in the Treatment of Fractures. EDWIN W. RYERSON, M.D., Chicago.

Thursday, 8:00 p.m.—Music Hall, Public Auditorium

LELAND S. MCKITTRICK, M.D., Boston; Vice President, American College of Surgeons, Presiding

Antibiotic and Chemotherapeutic Agents in Surgery

Pathologic Physiology of Surgical Infections. JOHN S. LOCKWOOD, M.D., New Haven

Penicillin—Its Use in Surgery and Influence on Earlier Types of Chemotherapy. WILLIAM A. ALTEMEIER, M.D., Cincinnati

Streptomycin. HORTON C. HINSHAW, M.D., Rochester, Minnesota

Correlation of the Use of Antibiotic and Chemotherapeutic Agents with General Principles of Surgery. S. CHAMP LYONS, M.D., New Orleans

OPHTHALMOLOGY

Symposia

Tuesday, 8:00 p.m.—South Hall A, Public Auditorium

F. BRUCE FRALICK, M.D., Ann Arbor; Professor of Ophthalmology, University of Michigan Medical School, Presiding

Orbital Reconstruction Including Prosthesis

Acrylic Prosthesis. A. D. RUEDEMANN, M.D., Cleveland

A New Type of Basket Implant for use after Enucleation. NORMAN L. CUTLER, M.D., Wilmington

Reconstruction of Orbital Floor Defects. Major ARTHUR E. SHERMAN, M.D., Springfield.

Reconstruction of the Upper Eyelid. EDMUND B. SPAETH, M.D., Philadelphia.

Thursday, 8:00 p.m.—South Hall A, Public Auditorium

ABRAM B. BRUNER, M.D., Professor of Ophthalmology, Western Reserve University School of Medicine, Presiding

Surgical Applied Anatomy in Eye Surgery. MEYER WIENER, M.D., Coronado.

Visual Disturbances Associated with Head Injuries. JOHN S. MCGAVIC, M.D., Fort Madison.

The Management of Non-Magnetic Intraocular Foreign Bodies. HARVEY E. THORPE, M.D., Pittsburgh.

The Management of Strabismus in Children. MAYNARD C. WHEELER, M.D., New York.

OTORHINOLARYNGOLOGY

Symposia

Tuesday, 8:00 p.m.—South Hall B, Public Auditorium

CARL H. MCCASKEY, M.D., Indianapolis; Member, Advisory Council for Otorhinolaryngology, American College of Surgeons, Presiding

Treatment of Deafness

The Management of the Chronic Suppurating Ear. JAMES H. MAXWELL, M.D., Ann Arbor.
The Fenestration Operation. GEORGE E. SHAMBAUGH, JR., M.D., Chicago.
The Use of Radium for Conductive Deafness. JOHN E. BORDLEY, M.D., Baltimore.

Thursday, 8:00 p.m.—South Hall B, Public Auditorium

Member Advisory Council for Otorhinolaryngology, American College of Surgeons, Presiding

Surgery of the Nasal Accessory Sinuses

Indications for Surgery in the Light of the Use of Antibiotics. HARRY P. SCHENK, M.D., Philadelphia
Intranasal Surgery. JOHN J. SHEA, M.D., Memphis.
External Operation. FRANCIS L. WEILLE, M.D., Boston.

PLASTIC SURGERY OF THE HEAD AND NECK

Symposium

Wednesday, 8:00 p.m.—South Hall A, Public Auditorium

GORDON B. NEW, M.D., Rochester, Minnesota; Member Advisory Council for Otorhinolaryngology, American College of Surgeons, Presiding

Cancellous Bone Grafts to the Jaw. TRUMAN G. BLOCKER, M.D., Galveston.
Repair of Nasal Defects with Free Composite Grafts of Skin and Cartilage from the Ear. JAMES B. BROWN, M.D., St. Louis.
Studies in the Anatomy and the Repair of Cleft Palate. GEORGE M. DORRANCE, M.D., Philadelphia.
Preservation of Function Following Resections of Jaw Tumors. LOUIS T. BYARS, M.D., St. Louis.

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

Tuesday through Friday, 9:00 a.m. to 12:30 p.m.—Music Hall, Public Auditorium

Presentation of the results of clinical and experimental research on problems related to general surgery and the surgical specialties which are being currently conducted in many medical schools, clinics, and hospitals.

SYMPOSIUM ON CANCER

Wednesday, 2:00 p.m.—Ballroom, Public Auditorium

FRANK E. ADAIR, M.D., New York; Chairman, Cancer Committee, American College of Surgeons, Presiding.

Surgical Treatment of Cancer of the Pancreas. RICHARD B. CATTELL, M.D., Boston.
Resections of Abdominal Cancer Infiltrating the Abdominal Wall. ALEXANDER BRUNSCHWIG, M.D., Chicago.
Hormone Therapy in Cancer of the Prostate. WILLIAM W. SCOTT, M.D., Baltimore.
Estrogen Therapy in Cancer of the Breast. IRA T. NATHANSON, M.D., Boston.
Testosterone in Cancer of the Breast. FRANK E. ADAIR, M.D., New York.
Observations on the Genetic Nature of Gastric Cancer in Mice. LEONELL C. STRONG, M.D., New Haven.

PANEL DISCUSSIONS

GENERAL SURGERY

Monday, 1:30–3:00 p.m.—Music Hall, Public Auditorium

Rehabilitation of the Surgical Patient and Early Ambulation

Moderator: HOWARD RUSK, M.D., New York
Collaborators: I. S. RAVDIN, M.D., Philadelphia
HENRY H. KESSLER, M.D., Newark
JOHN H. POWERS, M.D., Cooperstown

Monday, 3:30–5:00 p.m.—Music Hall, Public Auditorium

The Treatment of Cancer of the Large Bowel

Moderator: FREDERICK A. COLLER, M.D., Ann Arbor
Collaborators: THOMAS E. JONES, M.D., Cleveland
CLAUDE F. DIXON, M.D., Rochester, Minnesota
FRED W. RANKIN, M.D., Lexington

Tuesday, 1:30–3:00 p.m.—Music Hall, Public Auditorium

Thiouracil in Thyroid Disease

Moderator: FRANK LAHEY, M.D., Boston
Collaborators: GEORGE M. CURTIS, M.D., Columbus
WARREN H. COLE, M.D., Chicago
ELMER C. BARTELS, M.D., Boston

Tuesday, 3:30–5:00 p.m.—Music Hall, Public Auditorium

Recent Trends in the Management of Carcinoma of the Cervix

Moderator: JOE V. MEIGS, M.D., Boston
Collaborators: RICHARD W. TELINDE, M.D., Baltimore
CHARLES A. BEHNEY, M.D., Philadelphia
RICHARD DRESSER, M.D., Boston

Wednesday, 1:30–3:00 p.m.—Music Hall, Public Auditorium

Anesthesia

Moderator: HENRY K. BEECHER, M.D., Boston
Collaborators: URBAN H. EVERSOLE, M.D., Boston
LLOYD MOUSEL, M.D., Rochester, Minnesota
STUART C. CULLEN, M.D., Iowa City

Wednesday, 3:30–5:00 p.m.—Music Hall, Public Auditorium

Protein Metabolism in the Surgical Patient

Moderator: ROBERT ELMAN, M.D., St. Louis
Collaborators: CHARLES C. LUND, M.D., Boston
PAUL R. CANNON, M.D., Chicago
JOHN H. MULHOLLAND, M.D., New York

Thursday, 3:30–5:00 p.m.—Music Hall, Public Auditorium

Spinal Cord Injuries

Moderator: HOWARD C. NAFFZIGER, M.D., San Francisco
Collaborators: FRANCIS C. GRANT, M.D., Philadelphia
ROBERT H. KENNEDY, M.D., New York

Friday, 1:30–3:00 p.m.—Music Hall, Public Auditorium

Surgery of the Stomach

Moderator: ROSCOE R. GRAHAM, M.D., Toronto
Collaborators: G. GAVIN MILLER, M.D., Montreal
LESTER R. DRAGSTEDT, M.D., Chicago
HOWARD K. GRAY, M.D., Rochester, Minnesota

Friday, 3:30–5:00 p.m.—Music Hall, Public Auditorium

Surgery of the Vascular System

Moderator: DANIEL C. ELKIN, M.D., Atlanta
Collaborators: ALFRED BLALOCK, M.D., Baltimore
ARTHUR H. BLAKEMORE, M.D., New York
MICHAEL E. DEBAKEY, M.D., New Orleans

SURGICAL SPECIALTIES

Urology

Friday, 2:00–5:00 p.m.—South Hall C, Public Auditorium

Management of Spinal Cord Bladders

Moderator: HERMAN L. KRETSCHMER, M.D., Chicago
Collaborators: MICHAEL K. O'HEEREN, M.D., Houston. From the Standpoint of the Urologist
PHILIP LEWIN, M.D., Chicago. From the Standpoint of the Orthopedist
FRANK H. MAYFIELD, M.D., Cincinnati. From the Standpoint of the Neurologist
FRANK H. KRUSEN, M.D., Rochester, Minnesota. From the Standpoint of the Physical Therapist

Cancer of the Bladder—An Appraisal of Present Day Methods of Treatment

Moderator: HERMAN L. KRETSCHMER, M.D., Chicago
Collaborators: GERSHOM J. THOMPSON, M.D., Rochester, Minnesota. Conservative Transurethral Treatment of Bladder Tumors
F. H. SQUIRE, M.D., Chicago. Radiation Treatment
CHARLES C. HIGGINS, M.D., Cleveland. Cystectomy with Transplantation of the Ureters to the Bowel or Skin

Neurological Surgery

Friday, 2:00 to 5:00 p.m.—Club Room A, Public Auditorium

Ruptured Intervertebral Discs

Moderator: FRANCIS C. GRANT, M.D., Philadelphia
Collaborators: WILLIAM P. VANWAGENEN, M.D., Rochester, Minnesota
JAMES RUDOLPH JAEGER, M.D., Philadelphia
JAMES L. POPPEN, M.D., Boston

Orthopedic Surgery

Friday, 2:00–5:00 p.m.—Club Room B, Public Auditorium

Intra-Articular Fractures of the Elbow in Childhood and Adult Life

Moderator: CARL E. BADGLEY, M.D., Ann Arbor
Collaborators: PHILIP D. WILSON, M.D., New York
HAROLD B. BOYD, M.D., Memphis
CLAY R. MURRAY, M.D., New York

The Treatment of Club Foot and Its Complications

Moderator: CARL E. BADGLEY, M.D., Ann Arbor
Collaborators: JOSEPH H. KITE, M.D., Atlanta
FRANK R. OBER, M.D., Boston
FRANK CURTIS, M.D., Detroit

Plastic Surgery

Friday, 2:00–5:00 p.m.—Club Room C, Public Auditorium

Early Skin Grafting of Burns

Moderator: ROBERT H. IVY, M.D., Philadelphia
Collaborators: JEROME P. WEBSTER, M.D., New York
HARVEY S. ALLEN, M.D., Chicago

Use of Flaps and Grafts in Reconstructive Surgery of the Extremities
Moderator: ROBERT H. IVY, M.D., Philadelphia
Collaborators: Major CARL E. LISCHER, M.D., Valley Forge
Major WILLIAM B. DAVIS, M.D., Valley Forge

Obstetrics

Friday, 2:00–5:00 p.m.—South Hall A, Public Auditorium

Neonatal Morbidity and Mortality
Moderator: THADDEUS L. MONTGOMERY, M.D., Philadelphia
Collaborator: FREDERICK H. FALLS, M.D., Chicago

Thoracic Surgery

Friday, 2:00–5:00 p.m.—South Hall B, Public Auditorium

Treatment of Carcinoma of the Esophagus
Lung Resection in Pulmonary Tuberculosis
Moderator: RICHARD H. SWEET, M.D., Boston
Collaborators: JOHN M. GARLOCK, M.D., New York
WILLIAM E. ADAMS, M.D., Chicago
HERBERT C. MAIER, M.D., New York

OPHTHALMOLOGY

Tuesday, 11:00 a.m.—South Hall A, Public Auditorium

Retinal Detachment
Moderator: PAUL A. CHANDLER, M.D., Boston
Collaborators: DONALD W. BOGART, M.D., New York
PETER C. KRONFELD, M.D., Chicago

Wednesday, 11:00 a.m.—South Hall A, Public Auditorium

Glaucoma
Moderator: SAMUEL J. MEYER, M.D., Chicago
Collaborators: H. SAUL SUGAR, M.D., Chicago
THOMAS D. ALLEN, M.D., Chicago

Thursday, 11:00 a.m.—South Hall A, Public Auditorium

Keratoplasty
Moderator: R. TOWNLEY PATON, M.D., New York
Collaborators: DERRICK T. VAIL, JR., M.D., Chicago
HERBERT M. KATZIN, M.D., New York
J. WESLEY MCKINNEY, M.D., Memphis

OTORHINOLARYNGOLOGY

Tuesday, 11:00 a.m.—South Hall B, Public Auditorium

The Treatment of Meniere's Disease
Moderator: ALBERT C. FURSTENBERG, M.D., Ann Arbor
Collaborators: KENNETH M. DAY, M.D., Pittsburgh
JOHN R. LINDSAY, M.D., Chicago
THEODORE E. WALSH, M.D., St. Louis

Wednesday, 11:00 a.m.—South Hall B, Public Auditorium

Osteomyelitis of the Skull
Moderator: JOSEPH E. J. KING, M.D., New York
Collaborator: ALBERT C. FURSTENBERG, M.D., Ann Arbor

Thursday, 11:00 a.m.—South Hall B, Public Auditorium

Rehabilitation of the War Deafened
Moderator: NORTON CANFIELD, M.D., New Haven
Collaborators: FRANCIS L. LEDERER, M.D., Chicago
RAYMOND CARHART, Ph.D., Evanston
HERBERT KOEPP-BAKER, Ph.D., Chicago

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS
ADVANCES FROM WAR EXPERIENCE

Tuesday, 2:00 p.m.—Ballroom, Public Auditorium

ROBERT H. KENNEDY, M.D., New York; Chairman, Committee on Fractures and Other Traumas, Presiding.
Shock and Hemorrhage. CHARLES S. RIFE, M.D., Milwaukee.
Burns. Captain H. L. PUGH, Bethesda; M.C., U. S. Navy.
Injuries to the Chest. EARLE B. KAY, M.D., Cleveland.
Injuries to the Genitourinary Tract: Emphasis on Urethral Injuries. GEORGE C. PRATHER, M.D., Boston.
Injuries to the Rectum. EDMUND J. CROCE, M.D., Worcester.
Herniated Nucleus Pulposus. WINCHELL MCK. CRAIG, M.D., Rochester.
Spinal Cord Injuries. DAVID H. POER, M.D., Atlanta.
Peripheral Nerves. BENJAMIN B. WHITCOMB, M.D., Hartford.
Hand Surgery. WILLIAM H. FRACKELTON, M.D., Milwaukee.
Fractures of the Carpal Scaphoids. MATHER CLEVELAND, M.D., New York.
Transportation of Fractures. WILLIAM J. STEWART, M.D., Columbia.
Management of Compound Fractures in Their Early Phases. OSCAR P. HAMPTON, Jr., M.D., St. Louis
Osteomyelitis following Compound Fractures. GROVER C. PENBERTHY, M.D., Detroit.
Replacement of Skin Defects. JAMES B. BROWN, M.D., St. Louis.
Paravertebral Block and Sympathectomy. HARRIS B. SHUMACKER, Jr., M.D., Baltimore.
Bone Grafts. GEORGE K. CARPENTER, M.D., Nashville.
Amputations. RUFUS H. ALLDREDGE, M.D., New Orleans.
Training of Amputees. HENRY H. KESSLER, M.D., Newark.
Rehabilitation. LT. COL. A. WILLIAM REGGIO, Washington, D.C.
Brief Summary.
MAJOR GENERAL PAUL R. HAWLEY (Ret.), Washington; Chief Medical Director, U.S. Veterans Administration.
VICE ADMIRAL ROSS T. MCINTIRE, Washington; The Surgeon General of the Navy.
MAJOR GENERAL NORMAN T. KIRK, Washington; The Surgeon General of the Army.

GRADUATE TRAINING IN SURGERY AND THE SURGICAL SPECIALTIES
PANEL DISCUSSION

Thursday, 3:00 p.m.—Club Room B

From the standpoint of:
a. The American College of Surgeons
b. The Basic Medical Sciences
c. The Veterans Administration
d. The University-Connected Hospital
e. The Hospital Not University-Connected

ASSEMBLY OF INITIATES

Friday, 10:30 a.m.—Ballroom, Public Auditorium

IRVIN ABELL, M.D., Louisville; President, Presiding.
Opening Remarks—General Organization of the College.
IRVIN ABELL, M.D., Louisville.
The Program of the American College of Surgeons:
Hospital Standardization
Sectional Meetings
Medical Motion Pictures
Library and Literary Research
MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director and Chairman of the Administrative Board.

Graduate Training in Surgery
MAJOR GENERAL CHARLES R. REYNOLDS (Ret.), Chicago; Director, Department of Graduate Training in Surgery.
Credentials Department of Clinical Research; Cancer; Fractures and Other Traumas
BOWMAN C. CROWELL, M.D., Chicago; Associate Director and Vice Chairman of the Administrative Board.
Fellowship in the American College of Surgeons
ARTHUR W. ALLEN, M.D., Boston; Vice President, Board of Regents.
The Fellowship Pledge. Recital by Initiates.

ANNUAL MEETING, BOARD OF GOVERNORS OF THE AMERICAN COLLEGE OF SURGEONS

Wednesday, 12:00 Noon—Lattice Room, Hotel Statler

IRVIN ABELL, M.D., Louisville; President, Presiding.
Statement by the Chairman of the Board of Regents. IRVIN ABELL, M.D.
Brief Reports on the Activities, Problems and Progress of the American College of Surgeons.
BOWMAN C. CROWELL, M.D., Chicago; Associate Director and Vice Chairman of the Administrative Board.
MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director and Chairman of the Administrative Board.
DALLAS B. PHEMISTER, M.D., Chicago; Treasurer.
FREDERICK A. COLLER, M.D., Ann Arbor; Regent.
PAUL B. MAGNUSON, M.D., Chicago; Secretary.
Discussion by Governors and Regents.

ADJOURNED MEETING, BOARD OF GOVERNORS

Thursday, 1:30 p.m.—Music Hall, Public Auditorium

IRVIN ABELL, M.D., Louisville; Chairman of the Board of Regents, Presiding.
Report of Committee on Nominations to the Board of Governors.

ANNUAL MEETING, FELLOWS OF THE AMERICAN COLLEGE OF SURGEONS

Thursday, 1:45 p.m.—Music Hall, Public Auditorium

IRVIN ABELL, M.D., Louisville; President, Presiding.
Report of Committee on Nominations to the Fellows.
Financial Report.
DALLAS B. PHEMISTER, M.D., Chicago; Treasurer.
Report of Administrative Board.
MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director and Chairman of the Administrative Board.
Departmental Reports. a. Hospital Standardization. b. Sectional Meetings.
MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director.
Graduate Training in Surgery.
MAJOR GENERAL CHARLES R. REYNOLDS (Ret.), Chicago; Director, Department of Graduate Training in Surgery.
Credentials Department—Credentials Committees; Committees on Applicants; and Committee on History Reviews.
Clinical Research.
BOWMAN C. CROWELL, M.D., Chicago; Associate Director.
Medical Motion Pictures.
Publications.
ELEANOR K. GRIMM, Chicago; Administrative Executive.
Library and Literary Research.
L. MARGUERIETE PRIME, Chicago; Director of Library and Literary Research.
Public Relations.
LAURA G. JACKSON, Chicago; Director of Public Relations.

Committee on Cancer.
FRANK E. ADAIR, M.D., New York; Chairman.
Committee on Fractures and Other Traumas.
ROBERT H. KENNEDY, M.D., New York; Chairman.
Committee on Graduate Training in Surgery.
DALLAS B. PHEMISTER, M.D., Chicago; Chairman.
Fellowship Obligations and Opportunities.
ARTHUR W. ALLEN, M.D., Boston; Regent and Vice Chairman, Board of Regents.
Closing Remarks. The American College of Surgeons and Postwar Progress.
IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents.

COMMITTEE MEETINGS

IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents, Presiding.

STATE AND PROVINCIAL EXECUTIVE COMMITTEES

Wednesday, 9:30 a.m.—Ballroom, Hotel Statler

Statement by the Chairman of the Board of Regents.
IRVIN ABELL, M.D., Louisville.
Report on Sectional Meetings held in 1946 and Plans for Meetings in 1947.
MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director and Chairman of the Administrative Board.
Comments on Sectional Meetings, 1946–1947, by Chairmen of the Committees on Local Arrangements.
FRASER B. GURD, M.D., Montreal.
CHARLES S. KENNEDY, M.D., Detroit.
JAMES M. HAYES, M.D., Minneapolis.
HUGH TOLAND JONES, M.D., Los Angeles.
Discussion by the Fellows and Regents.

STATE AND PROVINCIAL CREDENTIALS COMMITTEES AND COMMITTEES ON APPLICANTS

Wednesday, 10:15 a.m.—Ballroom, Hotel Statler

Statement by the Chairman of the Board of Regents.
IRVIN ABELL, M.D., Louisville.
Report of Work of Credentials Committees and Committees on Applicants, 1946.
BOWMAN C. CROWELL, M.D., Chicago; Associate Director.
Interesting Observations in Interviewing Prospective Candidates.
H. PRATHER SAUNDERS, M.D., Chicago; Assistant Director.
The Junior Candidate.
MAJOR GENERAL CHARLES R. REYNOLDS (Ret.) Chicago; Director, Department of Graduate Training in Surgery.
Discussion by the Fellows and Regents.

STATE AND PROVINCIAL JUDICIARY COMMITTEES

Wednesday, 11:00 a.m.—Ballroom, Hotel Statler

Statement by the Chairman of the Board of Regents.
IRVIN ABELL, M.D., Louisville.
Selection and Appointment of Judiciary Committees and Procedure in Dealing with Judiciary Cases.
BOWMAN C. CROWELL, M.D., Chicago; Associate Director.
Types of Cases Referred to the Judiciary Committees.
MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director.
Discussion by Regents and Fellows of the College.

PRELIMINARY CLINICAL PROGRAM

CLEVELAND CITY HOSPITAL

Tuesday

General Surgery

S. O. FREEDLANDER, D. M. GLOVER, W. C. MCCALLY, M. B. TALIAK, J. H. LAZZARI, C. A. HAMANN, R. J. MCNAMEE, M. H. LAMBRIGHT, J. S. GELLER, A. M. LEIGH.

9:00 a.m.—Operative clinic.

Tumor Surgery

L. A. POMEROY, S. O. FREEDLANDER, H. H. HAUSER, J. H. LAZZARI, A. E. BENNETT, and staff.

2:00 p.m.—Tumor clinic. Case presentations for diagnosis and treatment.

Pathology

H. T. KARSNER, H. G. SCHLUMBERGER, R. C. TAYLOR, and staff.

2:00 p.m.—Clinical pathological conference.

Fractures and other traumas

F. M. BARRY, D. DIAL, and staff.

2:00 p.m.—Fracture service; ward rounds; discussions of treatment and results.

Thoracic Surgery

S. O. FREEDLANDER, R. C. MCKAY, M. C. PERLICH, C. W. MUNZ, D. B. KAY, and staff.

2:00 p.m.—Group conference. Selection of cases for thoracoplasty, lobectomy, and pneumonectomy.

Wednesday

General Surgery

S. O. FREEDLANDER, D. M. GLOVER, W. C. MCCALLY, M. B. TALIAK, J. H. LAZZARI, C. A. HAMANN, R. J. MCNAMEE, M. H. LAMBRIGHT, J. S. GELLER, A. M. LEIGH.

9:00 a.m.—Operative clinic.

2:00 p.m.—Nonoperative clinic:

M. H. LAMBRIGHT. The acute abdomen in patients carrying artificial pneumoperitoneum.

J. S. GELLER. Acute obstructive cholecystitis.

A. M. LEIGH. Esophageal diverticula.

W. C. MCCALLY. Vagus nerve resection for peptic ulcer.

J. H. LAZARRI. Hemipelvectomy for sarcoma of femur.

Plastic Surgery

D. M. GLOVER and C. A. HAMANN.

9:00 a.m.—Operative clinic.

2:00 p.m.—Nonoperative clinic; case presentations—problems and management of skin flaps. Lantern slides.

Otolaryngology

C. W. ENGLER, W. F. HULSE, S. E. MISSAL, CRAMER and Associates.

9:00 a.m.—Operative clinic.

2:00 p.m.—Nonoperative clinic; demonstration of oral and laryngeal malignancies.

Thursday

Neurosurgery

C. S. BECK, C. W. ELKINS, and associates.

9:00 a.m.—Operative clinic.

2:00 p.m.—Nonoperative clinic.

2:00 p.m.—C. W. ELKINS—Acrylic plastics in skull defects.

Vascular Surgery

L. A. ATLAS and associates.

9:00 a.m.—Operative clinic; lumbar sympathectomy; saphenous vein ligations.

2:00 p.m.—Nonoperative clinic: demonstration of venograms; indications for femoral vein ligations.

Genitourinary Surgery

H. R. TRATTNER, A. ROTH, and associates.

9:00 a.m.—Operative clinic.

General Surgery

R. W. SCOTT, S. O. FREEDLANDER, M. H. LAMBRIGHT, E. CHESTER, D. M. GLOVER, M. B. TALIAK, A. M. MARSHALL, J. H. LAZZARI, H. HOELSCHER, A. S. BROGLIO.

2:00 p.m.—Nonoperative clinic: lesions of stomach and intestine; pancreatic lithiasis with steatorrhea.

Friday

Thoracic Surgery

S. O. FREEDLANDER, D. B. KAY, C. W. MUNZ, and associates.

9:00 a.m.—Operative clinic: pneumonectomy, lobectomy, thoracoplasty.

2:00 p.m.—Nonoperative clinic.

2:00 p.m.—Motion pictures: esophagectomy, pneumonectomy, lobectomy.

Orthopedic Surgery

L. M. STARIN, A. TRAMER, P. H. DUBAY, and associates.

9:00 a.m.—Operative clinic: Spinal fusion.

9:00 a.m.—Nonoperative clinic.

Gynecology

J. L. REYCRAFT, A. E. BENNETT, E. RICHARDS, and associates.

9:00 a.m.—Operative clinic: Plastics; vaginal hysterectomy; Watkins' interposition.

Radiology

HARRY HAUSER and associates.

9:00 a.m.—A. R. COHEN—Roentgen diagnosis of lesions of small intestine.

10:00 a.m.—H. HAUSER—Roentgen diagnosis of thoracic tumors.

11:00 a.m.—B. BROWN—Differential roentgen diagnosis of mechanical and adynamic ileus.

CLEVELAND CLINIC

Tuesday

General Surgery

T. E. JONES and GEORGE CRILE, JR.
8:00 a.m.—Operative surgery and demonstration of cases.
R. L. HADEN, L. W. DIGGS, D. W. BORTZ and R. S. DINSMORE.
9:00 a.m.—Symposium—spleen: General considerations; platelets in hemorrhagic disease; presentation of cases; surgical aspects.
IRVINE H. PAGE, Leader; A. C. CORCORAN, ROBERT D. TAYLOR and W. JAMES GARDNER.
11:00 a.m.—Symposium—hypertension: General considerations; selection of cases for surgery; surgical aspects.

Genitourinary Surgery

CHARLES C. HIGGINS.
8:00 a.m.—Operative clinic.

Ophthalmology

A. D. RUEDEMANN.
8:00 a.m.—Operative clinic.

Orthopedic Surgery

JAMES A. DICKSON and J. I. KINDRICK.
8:30 a.m.—Operative clinic.

Otolaryngology

PAUL M. MOORE, JR.
10:00 a.m.—Operative clinic.
H. E. HARRIS.
1:00 p.m.—Operative clinic.

Pathology

JOHN B. HAZARD.
10:00 a.m.—Surgical pathological conference.
JOHN B. HAZARD.
11:45 a.m.—Demonstration of surgical pathological specimens.

Neurosurgery

W. JAMES GARDNER, A. T. BUNTS, and W. A. NOSIK.
1:00 p.m.—Operative clinic.

Wednesday

General Surgery

T. E. JONES.
8:00 a.m.—Operative clinic.
GEORGE CRILE, JR., Leader; E. N. COLLINS, JOHN F. RENSHAW, J. C. ROOT, and HAROLD R. ROSSMILLER.
9:00 a.m.—Symposium: Upper stomach and pancreas.
R. S. DINSMORE.
1:30 p.m.—Operative clinic.

Genitourinary Surgery

CHARLES C. HIGGINS.
8:00 a.m.—Operative clinic.
W. E. LOWER, CHARLES C. HIGGINS, W. J. ENGEL.
10:00 a.m.—Genitourinary clinic: Presentation of cases and discussion.
T. E. JONES and associates.
11:00 a.m.—Clinic: Diseases of colon.

Ophthalmology

A. D. RUEDEMANN and R. J. KENNEDY.
8:00 a.m.—Demonstration of cases.

Otolaryngology

PAUL M. MOORE, JR.
10:00 a.m.—Operative clinic and demonstration of cases.

Pathology

JOHN B. HAZARD.
11:45 a.m.—Demonstration of surgical pathological specimens.

Neurosurgery

W. JAMES GARDNER, A. T. BUNTS and W. A. NOSIK.
1:00 p.m.—Operative clinics.

Thursday

General Surgery

T. E. JONES and GEORGE CRILE, JR.
8:00 a.m.—Operative clinics.
IRVINE H. PAGE, Leader; A. C. CORCORAN, ROBERT D. TAYLOR, and W. JAMES GARDNER.
11:00 a.m.—Symposium—Hypertension: General considerations; selection of cases for surgery; surgical aspects.

Genitourinary Surgery

CHARLES C. HIGGINS.
8:00 a.m.—Operative clinic.
WILLIAM J. ENGEL.
1:00 p.m.—Operative clinic and demonstration of cases.

Ophthalmology

A. D. RUEDEMANN and R. J. KENNEDY.
8:00 a.m.—Operative clinic.

Orthopedic Surgery

JAMES A. DICKSON and J. I. KENDRICK.
8:30 a.m.—Operative clinic.
JAMES A. DICKSON, Leader; J. I. KENDRICK, and DR. PHALEN.
10:00 a.m.—Clinic—Hip joint.

Otolaryngology

PAUL M. MOORE, JR.
10:00 a.m.—Operative clinic.
H. E. HARRIS.
1:00 p.m.—Operative clinic.

Pathology

JOHN B. HAZARD.
11:45 a.m.—Demonstration of surgical pathological specimens.

Neurosurgery

W. JAMES GARDNER, Leader; A. T. BUNTS, W. A. NOSIK, and A. STOWELL.
9:00 a.m.—Clinic: Presentation of cases and discussion.
W. JAMES GARDNER, A. T. BUNTS, and W. A. NOSIK.
1:00 p.m.—Operative clinic.

Weekly Surgical Staff Conference

1:30 p.m.—Staff.

Friday

General Surgery

T. E. JONES.
8:00 a.m.—Operative clinics.
R. S. DINSMORE, E. PERRY MCCULLAGH, GEORGE CRILE, JR., and A. C. ERNSTENE.
9:00 a.m.—Thyroid clinic: Presentation of cases and discussion.
DONALD B. HALE, U. V. PORTMANN, and HAROLD E. HARRIS.
10:00 a.m.—Discussion of special problems: Safeguards in spinal anesthesia; radiation therapy in carcinoma of breast; treatment of esophageal varices.
IRVINE H. PAGE, Leader; A. C. CORCORAN, OTTO GLASSER, and W. JAMES GARDNER.
11:00 a.m.—Symposium—Shock: Crush syndrome; intra-arterial transfusions; induced hypotension.
R. S. DINSMORE.
1:30 p.m.—Operative clinic.

Genitourinary Surgery

CHARLES C. HIGGINS.
8:00 a.m.—Operative clinic.

Ophthalmology

A. D. RUEDEMANN, and R. J. KENNEDY.
8:00 a.m.—Operative clinic.

Orthopedic Surgery

JAMES A. DICKSON, and J. I. KENDRICK.
8:30 a.m.—Operative clinic.

Otolaryngology

PAUL M. MOORE, JR.
10:00 a.m.—Operative clinic.

Pathology

JOHN B. HAZARD.
11:45 a.m.—Demonstration of surgical pathological specimens.

Neurosurgery

W. JAMES GARDNER, A. T. BUNTS, and W. A. NOSIK.
1:00 p.m.—Operative clinic.

EVANGELICAL DEACONESS HOSPITAL

Wednesday

General Surgery

J. R. JOHNSON and associates.
9:00 a.m.—Operative and nonoperative clinic.

Thursday

General Surgery

O. A. WEBER and associates.
9:00 a.m.—Operative and nonoperative clinic.

Friday

General Surgery

S. C. LIND and associates.
9:00 a.m.—Operative and nonoperative clinic.

FAIRVIEW PARK HOSPITAL

Tuesday

General Surgery

W. E. SMITH and F. H. J. HEYSE.
9:00 a.m.—Operative clinic.

Obstetrics and Gynecology

E. D. SAUNDERS.
9:00 a.m.—Operative clinic.

Wednesday

General Surgery

H. W. MASENHIMER and J. R. KELKER.
9:00 a.m.—Operative clinic.

Genitourinary Surgery

L. F. HUFFMAN.
9:00 a.m.—Operative clinic.

Thursday

Orthopedic Surgery

G. I. BAUMAN.
9:00 a.m.—Operative clinic.

Roentgenology

E. V. BAEYER.
9:00 a.m.—Demonstration of films.

Obstetrics and Gynecology

E. D. SAUNDERS.
2:00 p.m.—Nonoperative clinic.

Friday

General Surgery

H. W. MASENHIMER, W. E. SMITH, J. R. KELKER, and F. H. J. HEYSE.
9:00 a.m.—Operative clinic.

Otolaryngology

W. H. RIEGER.
2:00 p.m.—Operative clinic.

GRACE HOSPITAL

Tuesday

Obstetrics and Gynecology

PAUL F. HASSE.
2:00 p.m.—Operative clinic.

Ophthalmology and Otolaryngology

H. W. COOPER.
2:00 p.m.—Operative clinic.

Wednesday

General Surgery

HARRY C. BARR.
9:00 a.m.—Operative and nonoperative clinic, rectal.
A. E. BIDDINGER.
2:00 p.m.—Operative clinic.

Thursday

General Surgery

F. P. GERACI.
9:00 a.m.—Operative clinic.
L. J. STERNICKI.
9:00 a.m.—Operative clinic.

Roentgenology

H. H. HAUSER.
2:00 p.m.—X-ray demonstration.

Friday

General Surgery

S. M. ADAMS.
9:00 a.m.—Operative clinic.

Genitourinary Surgery

C. B. MILLER.
9:00 a.m.—Operative genitourinary clinic.

Pathology

D. K. REHBOCK.
2:00 p.m.—Pathological demonstration.

HURON ROAD HOSPITAL

Tuesday

General Surgery

H. W. BROWN, J. L. BILTON, and W. J. RENNER.
8:00 a.m.—Gastric resections; intestinal resections.
R. J. WHITACRE and staff.
Anesthesia clinic.
E. A. MARSHALL.
2:00 p.m.—Nonoperative clinic—gastroenterology.

Obstetrics and Gynecology

A. G. CUMMINGS and G. H. IRVIN, JR.
8:00 a.m.—Operative clinic.

Wednesday

General Surgery

H. W. BROWN, J. L. BILTON, and W. J. RENNER.
8:00 a.m.—Thyroidectomies and mastectomies.
A. E. BIDDINGER.
2:00 p.m.—Surgical nonoperative clinic.
R. J. WHITACRE.
2:00 p.m.—Anesthesia clinic.
EDWARD GOODSITT.
2:00 p.m.—Operation of a blood bank in a general hospital.

Genitourinary Surgery

C. C. PERRY and L. K. BROOKS.
8:00 a.m.—Rectal surgery.
V. C. LAUGHLIN and departmental personnel.
2:00 p.m.—Nonoperative clinic: Urography and interpretation of pyelograms.

Obstetrics and Gynecology

G. H. IRVIN, JR., and G. J. SALISBURY.
8:00 a.m.—Vaginal hysterectomies and vaginal repairs.

Otolaryngology

G. H. QUAY.
9:00 a.m.—Operations: Harelip and mastoid.

Pathology

EDWARD GOODSITT.
2:00 p.m.—Clinicopathological conference.

Thursday

General Surgery

A. E. BIDDINGER, B. B. KIMMEL, T. L. LAUGHLIN, and L. L. CHANDLER.
8:00 a.m.—Operative clinic.
R. J. WHITACRE and staff.
2:00 p.m.—Anesthesia clinic.

Obstetrics and Gynecology

G. M. HAWK and S. E. BURKHART.
8:00 a.m.—Gynecological operations.

Genitourinary Surgery

V. C. LAUGHLIN and departmental personnel.
8:00 a.m.—Renal and prostatic surgery.

Orthopedic Surgery

A. H. REED and staff.
8:00 a.m.—Operative clinic: Fractured hips, open reductions, and tendon transplants.
A. H. REED and staff.
2:00 p.m.—Orthopedic nonoperative clinic.

Pathology

EDWARD GOODSITT.
2:00 p.m.—Clinicopathological conference.

Friday

General Surgery

H. W. BROWN, J. L. BILTON, and W. J. RENNER.
8:00 a.m.—Cholecystectomies and gastric resections.
R. J. WHITACRE and staff.
2:00 p.m.—Anesthesia clinic.

Obstetrics and Gynecology

S. E. BURKHART, G. H. IRVIN, JR., and A. G. CUMMINGS.
8:00 a.m.—Operative clinic.
A. G. CUMMINGS and G. H. IRVIN, JR.
2:00 p.m.—Nonoperative clinic.

Ophthalmology

JOSEPHINE DIRION PHILLIPS and C. I. THOMAS.
9:00 a.m.—Operations.

LAKESIDE HOSPITAL—UNIVERSITY HOSPITALS

Tuesday

General Surgery

DARRELL T. SHAW.
8:00 a.m.—Operative clinic: Plastic surgery.
CLAUDE S. BECK.
8:00 a.m.—Operative clinic: Neurosurgery and cardiac surgery.
CLAUDE S. BECK.
10:30 a.m.—Nonoperative clinic: Demonstration cardiac surgical cases.

Orthopedic Surgery

MAXWELL HARBIN, J. A. MURPHY, and W. J. MASSEY.
8:00 a.m.—Operative clinic.
10:30 a.m.—Demonstration of orthopedic cases.

Fractures and Other Traumas

DARRELL T. SHAW.
10:30 a.m.—Nonoperative clinic: Scar and wound excision.

Ophthalmology

A. B. BRUNER and LORAND JOHNSON.
8:30 a.m.—Operative clinic.

Pathology

HOWARD T. KARSNER.
12:30 p.m.—Pathological conference.

Wednesday

General Surgery

CARL H. LENHART, J. W. HOLLOWAY, FREDERICK R. MAUTZ, F. S. GIBSON, H. G. SLOAN, JOHN J. THORNTON, WILLIAM D. HOLDEN, FRANK M. BARRY, ELDEN C. WECKESSER.
8:00 a.m.—Operative clinics.
10:30 a.m.—Nonoperative clinics.
CARL H. LENHART. Introductory remarks.
FREDERICK R. MAUTZ. Surgical aspects of bleeding peptic ulcer.
JOHN W. HOLLOWAY. Ulcers of lesser curvature associated with hypoacidity.
FRANK M. BARRY. Summary of results obtained by Lakeside House officers in resection of the stomach.
WILLIAM D. HOLDEN. Treatment of venous thrombosis.
HIRAM O. STUDLEY. Percentage weight loss as a basic indicator of surgical risk in patients with chronic peptic ulcer.
JAMES J. JOELSON. Urinary calculi in recumbent patients.
FREDERICK R. MAUTZ. Esophagectomy for obstructive lesions of the esophagus.

Genitourinary Surgery

JAMES J. JOELSON and JOHN E. WILLIAMS.
8:00 a.m.—Operative clinics.

Thoracic Surgery

FREDERICK R. MAUTZ.
8:00 a.m.—Operative clinic.

Ophthalmology

A. B. BRUNER and M. P. MOTTO.
8:30 a.m.—Operative clinic.
C. I. THOMAS.
1:30 p.m.—Nonoperative clinic: Ophthalmological dispensary clinic.

Thursday

General Surgery

DARRELL T. SHAW.
8:00 a.m.—Operative clinic: Plastic surgery.
CLAUDE S. BECK.
8:00 a.m.—Operative clinic: Neurosurgery and cardiac surgery.
DARRELL T. SHAW.
10:30 a.m.—Nonoperative clinic: Application of tubes and flaps to surface defects of the extremities.
4:30 p.m.—Surgical conference.

Orthopedic Surgery

C. H. HEYMAN, W. G. MCGAW, and C. G. BARBER.
8:00-10:30 a.m.—Operative clinic.
10:30 a.m.-12:30 p.m.—Nonoperative clinic: Demonstration of orthopedic cases.

Ophthalmology

A. B. BRUNER and LORAND JOHNSON.
8:30 a.m.—Operative clinic.

Neurosurgery

CLAUDE S. BECK.
10:30 a.m.-12:30 p.m.—Demonstration of neurosurgical cases.

Friday

General Surgery

CARL H. LENHART, J. W. HOLLOWAY, FREDERICK R. MAUTZ, F. S. GIBSON, H. G. SLOAN, JOHN J. THORNTON, WILLIAM D. HOLDEN, FRANK M. BARRY, ELDEN C. WECKESSER.
8:00-10:30 a.m.—Operative clinics.
10:30 a.m.-12:30 p.m.—Nonoperative clinics:
WILLIAM D. HOLDEN. Surgical treatment of arteriosclerotic and diabetic gangrene.
FRANK M. BARRY. Presentation of two cases of adenoma of the pancreas.
JOHN W. HOLLOWAY. Adenoma of parathyroid with hyperparathyroidism (7 cases).
JOHN J. THORNTON. An unusual case of esophageal diverticulum.
ELDEN C. WECKESSER. Surgery of the hand.
JOHN E. WILLIAMS. Treatment of perirenal and renal abscesses.

Genitourinary Surgery

JAMES J. JOELSON and JOHN E. WILLIAMS
8:00 a.m.—Operative clinic.

Ophthalmology

A. B. BRUNER and C. I. THOMAS.
8:30 a.m.—Operative clinic.
W. B. CHAMBERLIN, JR.
1:30 p.m.—Nonoperative clinic: Ophthalmological dispensary clinic.

LAKEWOOD HOSPITAL

Tuesday

Obstetrics and Gynecology

BURDETT WYLIE.
2:00 p.m.—Nonoperative clinic: Discussion of obstetrical problems in a small general hospital.

Otolaryngology

Staff.
2:00 p.m.—Nonoperative clinic: Peroral endoscopy; vertigo due to eustachian tube obstruction and its treatment.

Genitourinary Surgery

L. F. HUFFMAN.
2:00 p.m.—Nonoperative clinic: Urological demonstrations.

Wednesday

General Surgery

C. LEE GRABER and associate members of visiting surgical staff.
9:00 a.m.—Operative clinics.
C. LEE GRABER.
2:00 p.m.—Nonoperative clinic: Improved and simplified technique for intravenous medication.

Orthopedic Surgery

LOUIS M. STARIN and PAUL H. DUBE.
9:00 a.m.—Operative and nonoperative clinic.

Otolaryngology

FORREST W. MERICA.
2:00 p.m.—Nonoperative clinic: Vertigo due to eustachian tube obstruction and its treatment.

Thursday

General Surgery

NORTH W. SHETTER.
9:00 a.m.—Exhibit of interesting x-ray cases; discussion of radiological problems.
STANLEY N. P. DEVILLE and G. C. RANKIN.
9:00 a.m.—Demonstration of technique of spinal anesthesia.

Otolaryngology

RALPH W. FRACKELTON.
9:00 a.m.—Operations: Nasal plastic.
Staff.
2:00 p.m.—Nonoperative clinic: Peroral endoscopy.

LUTHERAN HOSPITAL

Tuesday

General Surgery

D. S. SPRENG, J. H. LAZZARI, F. A. CATALANO, W. R. HODGES, H. H. ROENICK, E. R. HEXTER.
9:00 a.m.—Operative clinic.
J. P. EICHHORN, J. A. GROH, and H. G. SLOAN.
10:00 a.m.—Nonoperative clinic—Symposium on peptic ulcer: Medical aspects; radiological aspects; surgical aspects.

Wednesday

General Surgery

H. G. SLOAN, B. B. LARSEN, V. L. TICHY, J. H. TILDES, E. C. ROY, J. F. HATTENBACH.
9:00 a.m.—Operative clinic.
Staff—J. H. LAZZARI, Chairman.
10:30 a.m.—Tumor clinic—Presentation of current cases.

Thursday

General Surgery

F. S. GIBSON, J. T. BOHM, G. A. TISCHLER, C. E. WARD C. F. WARD, CONRAD OTTELIN, C. W. HAHN.
9:00 a.m —Operative clinic.
F. S. GIBSON, FRANCIS BAYLESS, and J. A. GROH.
10:30 a.m.—Nonoperative clinic: Symposium on diseases of the breast: The clinical diagnosis; pathology of breast tumors; the value of x-ray as a postoperative agent.

Friday

General Surgery

S. C. LIND, J. H. LAZZARI, I. J. GOODMAN, B. B. LARSEN, D. S. SPRENG.
9:00 a.m.—Operative clinic.

Ophthalmology

W. P. CHAMBERLAIN, JR.
9:00 a.m.—Operative clinic.

Genitourinary Surgery

D. A. CHAMBERS and W. E. FORSYTHE.
9:00 a.m.—Operative clinic.

THE POLYCLINIC HOSPITAL

Tuesday

General Surgery

HENRY A. SCHLINK.
8:00 a.m.—Operative clinic.
JOHN R. PLENT.
10:00 a.m.—Operative clinic.

Obstetrics and Gynecology

WILLIAM N. MACEY.
11:00 a.m.—Operative clinic.

Wednesday

General Surgery

PAUL M. SPURNEY.
9:00 a.m.—Operative clinic.
WILLIAM KUTLER.
10:00 a.m.—Operative clinic.
CHARLES J. PROCHASKA.
11:00 a.m.—Operative clinic.

Fractures and Other Traumas

S. L. ROBBINS.
9:00 a.m.—Fracture clinic.

Genitourinary Surgery

HARRY R. TRATTNER.
9:00 a.m.—Nonoperative clinic: Prostatectomy.

Thursday

General Surgery

JOHN T. VITKUS.
9:00 a.m.—Operative clinic.
A. J. KAZLAUCKAS.
10:00 a.m.—Operative clinic.

Otolaryngology

A. L. STOTTER.
9:00 a.m.—Nasal reconstruction.

Pathology

Nonoperative clinic.
11:00 a.m.—Clinical pathological conference.

Friday

Nonoperative clinic.
9:00 a.m.—Carcinoma, corpus uteri; spontaneous rupture of uterus; intestinal obstruction; ectopic gestation.

MacDONALD HOUSE—UNIVERSITY HOSPITALS

Tuesday

Gynecology and Obstetrics

J. L. Reycraft.
9:30 a.m.—Gynecology operative clinic.
W. R. Barney.
11:00 a.m.—Ward rounds.

Wednesday

Gynecology and Obstetrics

R. L. Faulkner.
9:30 a.m.—Symposium: Uterine bleeding.
L. A. Pomeroy.
11:00 a.m.—Carcinoma cervix.

Thursday

Gynecology and Obstetrics

R. L. Faulkner.
9:30 a.m.—Obstetrics and gynecology pathological conference.
R. A. Shipley.
11:00 a.m.—Presentation of gynecological endocrine problems.

MOUNT SINAI HOSPITAL

Tuesday

Orthopedic Surgery

Rudolph S. Reich, A. Miller, Tramer, Klein, Driehls.
8:00 a.m.—Operative clinic.

Obstetrics and Gynecology

Jacob L. Bubis and staff.
9:00 a.m.—Nonoperative clinic.

Oral Surgery

Edward Reiter.
2:00 p.m.—Nonoperative clinic.

Genitourinary Surgery

D. Schneider.
9:00 a.m.—Nonoperative clinic: Traumatic-urological complications of abdominal surgery.
J. Galvin.
9:00 a.m.—Nonoperative clinic: Carcinoma of the ureter.
A. Roth.
9:00 a.m.—Nonoperative clinic: Carcinoma of the kidney.

Wednesday

General Surgery

S. O. Freedlander, H. M. Gans, Pevaroff, Hoffman, Geller, Bookatz.
8:00 a.m.—Operative clinic.

Pathology

Doctors Kline and Young.
2:00 p.m.—Clinical pathology.

Ophthalmology

H. M. Gans and staff.
2:00 p.m.—Nonoperative clinic: Ophthalmological problems illustrated with kodachrome slides.

Otorhinolaryngology

S. S. Quittner and S. Baumoel.
9:00 a.m.—Nonoperative clinic: Vertigo.

Thursday

Obstetrics and Gynecology

Jacob L. Bubis and staff.
8:00 a.m.—Operative clinic.

General Surgery

S. O. Freedlander and staff.
9:00 a.m.—Nonoperative clinic.

Pathology

Doctors Kline and Young.
2:00 p.m.—Pathological conference.

Friday

General Surgery

S. O. Freedlander and staff.
8:00 a.m.—Operative clinic.

Orthopedic Surgery

Rudolph S. Reich and staff.
9:00 a.m.—Nonoperative clinic: End results of ununited fractures of the femoral neck; fractures of the elbow; fractures of the calcaneous; unreduced Pott's fractures.

ST. ALEXIS HOSPITAL

Tuesday

General Surgery

J. F. Corrigan, W. F. Boukalik, E. F. Kieger, E. A Mastics, F. A. Spittler, J. N. Wychgel.
9:00 a.m.—Operative clinic.

Genitourinary Surgery

J. A. Sommer and C. R. Nuckolls.
9:00 a.m.—Operative clinic.

Neurosurgery

E. W. Shannon.
9:00 a.m.—Operative clinic.

Ophthalmology

H. V. Phelan.
9:00 a.m.—Operative clinic.
10:00 a.m.—Demonstration of cases.

Otolaryngology

F. W. Dixon and Fred Adelstein.
9:00 a.m.—Operative clinic.

Wednesday

General Surgery

J. F. Corrigan, W. F. Boukalik, E. F. Kieger, E. A. Mastics, F. A. Spittler, J. N. Wychgel.
9:00 a.m.—Operative clinic.

Genitourinary Surgery

J. A. SOMMER and C. R. NUCKOLLS.
9:00 a.m.—Operative clinic.

Ophthalmology

H. V. PHELAN.
9:00 a.m.—Operative clinic.

Thursday

General Surgery

J. F. CORRIGAN, W. F. BOUKALIK, E. F. KIEGER, E. A. MASTICS, F. A. SPITTLER, J. N. WYCHGEL.
9:00 a.m.—Operative clinic.

Genitourinary Surgery

J. A. SOMMER and C. R. NUCKOLLS.
9:00 a.m.—Operative clinic.

Neurosurgery

E. W. SHANNON.
9:00 a.m.—Operative clinic.

Ophthalmology

H. V. PHELAN.
9:00 a.m.—Operative clinic.

Otolaryngology

F. W. DIXON and FRED ADELSTEIN.
9:00 a.m.—Operative clinic.

ST. JOHN'S HOSPITAL

Tuesday

General Surgery

J. R. RIPTON.
9:00 a.m.—Thyroid surgery.
J. W. HANNIBAL.
9:00 a.m.—Gall-bladder and common duct surgery.
WILLIAM NOVINCE.
9:00 a.m.—Discussion of liver function tests.

Otolaryngology

J. T. COLLINS.
9:00 a.m.—Radical antrum.

Neurosurgery

E. W. SHANNON.
9:00 a.m.—Intervertebral disc.

Wednesday

General Surgery

J. R. RIPTON.
9:00 a.m.—Gall bladder.
C. E. STEYER.
11:00 a.m.—Nonoperative clinic—liver and gall bladder.
WILLIAM MISHLER.
Resection of right half of colon for carcinoma of cecum.

Genitourinary Surgery

J. A. SOMMER.
9:00 a.m.—Nephrectomy.
WILLIAM J. MANNING.
10:00 a.m.—Indications for suprapubic prostatectomy.

Roentgenology

DAVID STEEL.
2:00 p.m.—Tumor clinic.
H. C. HARTZELL.
2:00 p.m.—Tumor clinic.

Thursday

General Surgery

F. T. GALLAGHER.
9:00 a.m.—Gastric resection (indications for Billroth No. 1 and Hoffmeister-Balfour resections).
J. E. HANNIBAL.
9:00 a.m.—Thyroid surgery.

Fractures and Other Traumas

R. J. SCHRAFF.
10:00 a.m.—Intracapsular fracture of neck of femur, Smith-Petersen nail.

Otolaryngology

F. L. MCGANNON.
10:00 a.m.—Hemangioma of uvula. (Nonoperative)

Thoracic Surgery

C. W. MUNZ.
11:00 a.m.

Friday

General Surgery

J. E. HANNIBAL.
9:00 a.m.—Carcinoma of the recto-sigmoid, anterior resection.
F. T. GALLAGHER.
9:00 a.m.—Combined abdomino-perineal resection.
R. J. SCHRAFF.
9:00 a.m.—Skin graft. (Nonoperative)
WILLIAM E. MISHLER.
11:00 a.m.—Injury to knee.

Otolaryngology

DAVID STEEL.
10:00 a.m.—Radiation in otolaryngology.

ST. LUKE'S HOSPITAL

Tuesday

General Surgery

D. M. GLOVER, R. S. MCGINNIS, and staff.
9:00 a.m.—Operative clinic.
B. B. SANKEY and staff.
9:00 a.m.—Demonstrations in anesthesia.

Thoracic Surgery

EARLE B. KAY.
9:00 a.m.—Operative clinic.

Pathology

RAFAEL DOMINGUEZ and staff.
1:30 p.m.—Surgical pathological conference.

General Surgical Clinic

ERNEST F. BRIGHT.
2:30 p.m.—Peripheral vascular clinic.
R. S. MCGINNIS.
2:50 p.m.—Early ambulation in relation to postoperative complications.

HAROLD J. THEISEN.
3:10 p.m.—Amino acid therapy.
EDMUND E. BEARD.
3:20 p.m.—Thiouracil in thyrotoxicosis.
JOHN A. TOPINKA.
3:50 p.m.—Work of the tissue committee.
JOHN H. NICHOLS.
4:00 p.m.—Psychic disorders simulating surgical conditions.
D. M. GLOVER.
4:20 p.m.—Wound management.
Discussion.
4:35 p.m.

Thoracic Surgical Clinic

EARLE B. KAY.
2:30 p.m.—Pneumonectomies (motion pictures).
EARLE B. KAY
2:50 p.m.—Esophageal resections for stricture.
A. D. NICHOL.
3:10 p.m.—Follow-up on patent ductus cases from the cardiac clinic.
Discussion.
3:40 p.m.

Wednesday

General Surgery

B. B. SANKEY and staff.
9:00 a.m.—Demonstrations in anesthesia.

Neurosurgery

SPENCER BRADEN.
9:00 a.m.—Operative clinic.
2:00 p.m.—Neurosurgical clinic:
SPENCER BRADEN.
2:00 p.m.—The herniated disc.
DON D. BRANNAN.
2:30 p.m.—Myelography.
F. R. HILL and SPENCER BRADEN.
2:50 p.m.—Management of head injuries.
JOHN H. NICHOLS.
3:00 p.m.—The traumatic neurosis.

Orthopedic and Traumatic Surgery

T. A. WILLIS and staff.
9:00 a.m.—Operative clinic.
3:30 p.m.—Orthopedic and traumatic surgery clinic:
T. A. WILLIS.
3:30 p.m.—Management of the fractured hip.
B. B. LARSEN.
3:50 p.m.—Fixation of the hip (motion pictures).
W. H. MCGAW.
4:10 p.m.—Internal derangements of the knee; pneumograms.
Discussion.
4:40 p.m.

Obstetrics and Gynecology

C. T. HEMMINGS, G. B. HURD, and staff.
9:00 a.m.—Operative clinic.
2:00 p.m.—Obstetrical and gynecological clinic:
C. T. HEMMINGS.
2:00 p.m.—Conduct of normal delivery (motion pictures).
C. T. HEMMINGS.
2:30 p.m.—Operative obstetrics (motion pictures).
GERALD B. HURD.
3:00 p.m.—Management of uterine displacements.

Tumor Conference

D. D. BRANNAN and R. DOMINGUEZ.
3:30 p.m.—Lymphoma.
L. A. POMEROY.
3:45 p.m.—Carcinoma of the cervix.
GERALD B. HURD.
4:00 p.m.—Carcinoma of the uterine body.
R. B. HERRICK.
4:15 p.m.—Bone tumors.
R. S. MCGINNIS.
4:30 p.m.—Carcinoma of stomach; carcinoma of colon.
P. J. ROBECHEK.
4:45 p.m.—Carcinoma of the breast.

Thursday

General Surgery

B. B. SANKEY and staff.
9:00 a.m.—Demonstrations in anesthesia.
2:00 p.m.—Clinic on anæsthesiology:
G. R. HAMILTON.
2:00 p.m.—Supportive therapy in the operating room.
R. M. CRANE.
2:20 p.m.—Anesthesia in thyroid surgery.
L. E. CAMPBELL.
2:40 p.m.—Clinical aspects of curare.
B. B. SANKEY.
3:00 p.m.—Some aspects of spinal anesthesia.

Plastic and Maxillofacial Surgery

D. M. GLOVER and D. T. SHAW.
9:00 a.m.—Operative clinic.
D. M. GLOVER, D. T. SHAW, C. A. HAMANN, and GAYLORD JAMES, D.D.S.
3:15 p.m.—Plastic and maxillofacial surgery clinic: operative management of the harelip (motion pictures); Cleft Palate Problems: surgery, speech training, prosthesis; various applications of split thickness grafts; pedicle grafts; hand problems; reconstruction of external ear; temporomandibular ankylosis.

Orthopedic and Traumatic Surgery

T. A. WILLIS.
9:00 a.m.—Orthopedic and traumatic surgical operations.
WALLACE S. DUNCAN, T. A. WILLIS, W. A. ENGEL, D. M. GLOVER.
2:00 p.m.—Orthopedic and traumatic surgery clinic: Disabilities of the ankle joint; low back pain; 20 year review of burns.

Friday

General Surgery

B. B. SANKEY and staff.
9:00 a.m.—Anesthesia demonstrations.
9:00 a.m.—General surgical clinic.
P. J. ROBECHEK.
9:00 a.m.—Postoperative complications and deaths.
R. S. MCGINNIS.
9:15 a.m.—Intestinal obstruction.
B. B. LARSEN.
9:35 a.m.—The pilonidal sinus; operative treatment (motion pictures).
W. C. FARGO.
10:00 a.m.—Pyloric stenosis in the infant.
D. M. GLOVER.
10:20 a.m.—Anomalies of the intestinal tract in the newborn.

J. G. JONES.
10:45 a.m.—Biliary fistula—subphrenic abscess—bronchial fistula.
Discussion.
11:00 a.m.

Genitourinary Surgery

C. A. BOWERS and staff.
9:00 a.m.—Urological operations.
2:00 p.m.—Urological clinic.
D. A. CHAMBERS.
2:00 p.m.—Nephrectomy in surgical hypertension.
H. B. WRIGHT.
2:30 p.m.—Treatment of vesicovaginal fistula.
JAMES LYNCH.
3:00 p.m.—Cysts of the kidney.
D. W. MCINTYRE.
3:30 p.m.—Discussion of renal tumors.
C. A. BOWERS.
4:00 p.m.—The present status of prostatic surgery.

Otorhinolaryngology

THOMAS W. THOBURN and staff.
9:00 a.m.—Otorhinolaryngological operations.
2:00 p.m.—Otorhinolaryngological clinic.
J. W. MCCALL.
2:00 p.m.—The esophageal voice in laryngectomized patients.
C. E. KINNEY.
2:30 p.m.—Results of the fenestration operation.
F. W. ALEXANDER.
3:00 p.m.—Common errors in rhinoplasty.
T. W. THOBURN.
3:30 p.m.—Management of the nasal septum tip deflection in rhinoplasty.
WARD MILLS.
4:00 p.m.—Case of double malignancy of the larynx.
LEE STONER.
4:15 p.m.—Esophageal stricture due to lye ingestion, complicated by peri-esophageal metastases of a suprarenal tumor.

Ophthalmology

M. W. JACOBY and staff.
9:00 a.m.—Ophthalmological clinic.
PAUL G. MOORE.
9:00 a.m.—Traumatic eye surgery: intraocular foreign bodies.
M. W. JACOBY.
9:30 a.m.—Traumatic eye surgery: contusions of globe, intraocular hemorrhage and secondary glaucoma.
Discussion.
10:00 a.m.
GERALD T. SCHWARZ and M. I. SPARKS.
10:15 a.m.—The albumin-globulin ratio: its relation to diabetic retinopathy.
B. J. WOLPAW.
10:45 a.m.—Visual requirements for flying (original study, Naval Flying School, Pensacola).
Discussion.
11:15 a.m.
E. F. HARVEY.
11:30 a.m.—Interesting fundus cases: a. Pits of optic nerve; b. Division of optic nerve with extensive field defect; c. Angioid streaks of retina.
Discussion.
12:00 noon.

ST. VINCENT CHARITY HOSPITAL

Tuesday

Ophthalmology

H. H. WYAND and associates.
2:00 p.m.—Nonoperative clinic.

Wednesday

General Surgery

O. A. WEBER and associates.
9:00 a.m.—Operative clinic.

Orthopedic Surgery

C. G. BARBER and associates.
9:00 a.m.—Operative clinic.

Thursday

General Surgery

Tumor clinic.
8:00 a.m.
J. E. HALLISY and associates.
9:00 a.m.—Operative clinic.

Otolaryngology

C. W. ENGLER and associates.
9:00 a.m.—Operative clinic.

Friday

General Surgery

E. P. NEARY and associates.
9:00 a.m.—Operative clinic.

Genitourinary Surgery

H. R. TRATTNER, L. F. HUFFMAN, and H. R. WRIGHT.
9:00 a.m.—Operative clinic.

UNITED STATES MARINE HOSPITAL

Tuesday

Genitourinary Surgery

D. W. MCINTYRE.
9:00 a.m.—Clinic, followed by operations.

Orthopedic Surgery

T. A. WILLIS.
9:00 a.m.—Nonoperative clinic.